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Loci of Signal Probability Effects and of the Attentional Blink Bottleneck

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To investigate the locus of signal probability effects and the influence of stimulus quality on this locus, the authors manipulated probability in Task 2 of a psychological refractory period (PRP) paradigm. The effect was additive with stimulus onset asynchrony (SOA) when the target was not masked but underadditive with decreasing SOA when the target was masked. Even with masking, however, a range of probabilities had effects additive with SOA. The results suggest loci of stimulus probability before the PRP bottleneck as well as at or after the bottleneck. A second issue addressed was the locus of interference in the attentional blink (AB). The AB was larger when the probability of the first of 2 targets was lower. The results lead to the conclusion that one cause of the AB effect is a locus at least as late as the PRP bottleneck.

This article reports two sets of empirical investigations that had both separate and interrelated goals. In the first set of experiments (Experiments 1–3), we used the psychological refractory period (PRP) paradigm to constrain the locus of signal probability manipulations in simple choice tasks. These experiments and a selective review of the literature suggest that there are a range of signal probabilities that affect primarily central stages of processing. In the second set of studies (Experiments 4 and 5), we measured the effects of signal probability manipulations in the context of the speeded attentional blink (SAB) paradigm (Experiment 4) and the attentional blink (AB) paradigm (Experiment 5). These experiments showed that manipulations of the signal probability of the first target (T_1) had a large impact on the magnitude of the deficit in reporting the second target (T_2), lower signal probabilities in Task 1 caused larger deficits in Task 2. The overarching goal was to use what we learned about signal probability in the PRP experiments to constrain the locus of dual-task interference (which we call the bottleneck) in the SAB and AB paradigms. The results and our analysis suggest that at least some of the SAB and AB effects occur relatively late in the information-processing stream. The theoretical analysis and logic required to arrive at this conclusion are outlined in subsequent sections.

The Attentional Blink

When rapid serial visual presentation (RSVP) of stimuli is used, one often observes interference and confusion between sequential items. If observers are asked to look for two targets in such presentations, the proportion of correct identifications of T_2 generally decreases as the number of distractor items between T_1 and T_2 is reduced. This attenuation in accurately identifying T_2 when it appears shortly after T_1 is called the AB (Raymond, Shapiro, & Arnell, 1992). The AB effect has been shown to generalize to many types of T_2 stimuli, including letters (Raymond et al., 1992), colors (Ross & Jolicœur, 1999), words (Broadbent & Broadbent, 1987), orientation (Joseph, Chun, & Nakayama, 1997), and random dot patterns (Shapiro, Raymond, & Arnell, 1994, Experiment 4).

Evidence suggests that interference effects such as AB are a result of attentional limitations in people's ability to process information (Chun & Potter, 1995; Jolicœur, 1998; Luck, Vogel, & Shapiro, 1996; Raymond et al., 1992). However, our understanding of the nature of these limitations is incomplete, and more work is needed to pinpoint the processing stage(s) at which interference occurs. The goal of this article is to provide additional constraints on the possible locus, or loci, of the AB phenomenon.

Several theories purport to describe the fundamental mechanism underlying the AB effect (Chun & Potter, 1995; Duncan, Ward, & Shapiro, 1994; Jolicœur, 1998; Raymond et al., 1992; Shapiro et al., 1994). Although there is a general consensus among researchers that the AB effect is postperceptual (Luck et al., 1996), more work will be required to pinpoint the locus and nature of the interference that leads to the effect. In this study, we manipulated the relative probability of T_1 in the AB paradigm and observed large changes in the magnitude of the AB effect. These results provide new constraints on theories of the phenomenon.

The Psychological Refractory Period

We used the PRP paradigm in this study as a new tool to investigate signal probability effects. Our goal was to discover the locus of signal probability effects, relative to the processing bottleneck in the PRP paradigm, using manipulations that could also be used in the context of the AB paradigm, because one of our aims was to create links between the two paradigms. This section describes the PRP effect and reviews the literature, providing evidence for a postponement account for the effect with a locus at or before the response selection stage of processing.

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The PRP effect is observed as a slowing in performance on a second task (Task 2) as a function of its temporal proximity to a first task (Task 1). Response times (RTs) to Task 2 are typically delayed when stimuli are presented at shorter stimulus onset asynchronies (SOAs) relative to when they are presented at longer SOAs (Pashler, 1994a, Welford, 1952). Found even when very simple tasks, such as simple RT, are used, the PRP effect is a robust interference effect that is often relatively resistant to practice (Gottsdanker & Stelmach, 1971, McCann & Johnston, 1992, Van Selst & Jolicœur, 1997; but see Van Selst, Ruthruff, & Johnston, 1999). It has been observed with visual stimuli (Luck, 1998, Pashler, 1994b) as well as under cross-modal conditions (McCann & Johnston, 1992; Pashler, 1994a, Van Selst & Jolicœur, 1997). Moreover, output modality does not need to be the same for each task. For example, Pashler (1989, 1990) observed PRP effects when manual and vocal responses were combined.

The most widely accepted account of the fundamental mechanism underlying dual-task interference is that of postponement or delay in processing (Pashler, 1994a; Welford, 1952) caused by a processing bottleneck. In this view, the bottleneck mechanisms can work on only one information-processing stream at a time. Consequently, if these mechanisms are occupied by processing required to complete Task 1, processing for Task 2 must wait. It is this period of waiting, or period of cognitive slack (McCann & Johnston, 1992, Pashler, 1984, Pashler & Johnston, 1989), that is reflected in the slowing of RT to the second task as SOA is reduced. We call this class of models *all-or-none bottleneck models*. An alternate account of dual-task interference is provided by central capacity sharing models (Tombu & Jolicœur, 2001). This class of models has as a key assumption the notion that central resources can be shared when stimuli appear in close succession to each other, and the effect of this division is seen as a slowing in RT. For present purposes, however, both classes of models make the same predictions. Hence, we do not attempt to distinguish between them, and we refer to central capacity limitations broadly as *the bottleneck* while remaining neutral as to the detailed nature of that bottleneck (all-or-none vs. graded capacity sharing). Given that the key predictions logically needed later in this article can be derived from both classes of models, we use the now standard locus-of-slack logic to describe them (but see Tombu & Jolicœur, 2001, for a different way to arrive at the same predictions).

The locus of cognitive slack logic provides a means of interpreting the effects of factor manipulations in the PRP paradigm so as to constrain their locus in the information-processing stream (McCann & Johnston, 1992, Pashler & Johnston, 1989). We are interested in two of the major predictions from this type of model: (a) additivity of SOA and of manipulations with an effect at or after the bottleneck and (b) carry forward to Task 2 of a Task 1 bottleneck or prebottleneck manipulation. We consider each of these two predictions in more detail in the following paragraphs.

Figure 1 depicts the stages of processing necessary to perform two tasks with varying SOAs. In this hypothetical example, the bottleneck stages are labeled B. By assumption, Stages 1B and 2B cannot occur in parallel. If the Task 2 manipulation has a locus at the bottleneck (Stage 2B) or any subsequent stage, the effect will be seen as a slowing in mean RT to Task 2. Furthermore, the slowing will be consistent across SOAs, because the effect of the manipulation occurs after processing resumes, and therefore the slowing caused by the manipulation delays the output of the response by the same amount regardless of SOA. Thus, the model predicts additivity of this manipulation and SOA. We contrast this prediction with another interesting prediction of this type of model, namely the predicted underadditivity of SOA and a prebottleneck Task 2 manipulation. If the manipulation has an effect before the bottleneck, at Stage 2A in Figure 2, additional processing due to the difficulty manipulation will be absorbed in the period of cognitive slack if the SOA is short. However, the full effect will still be observed at long SOAs because there is no postponement of central stages in Task 2 and thus no slack to absorb the effect of the manipulation. Thus, the effect, as seen in RT to Task 2, will be underadditive with decreasing SOA (the difficulty effect decreasing as SOA decreases).

Manipulating a variable that is likely to have an effect at a specific stage of processing (e.g., sensory encoding or response selection) and interpreting the pattern of data according to the locus of cognitive slack logic just described (McCann & Johnston, 1992, Pashler & Johnston, 1989) is a useful method for obtaining empirical evidence regarding the locus (or loci) of the PRP bottleneck. Much of the evidence as to the locus of the PRP effect points to a relatively late stage of processing (McCann & Johnston, 1992, Schubert, 1999, Van Selst & Jolicœur, 1997). Evidence against an early, perceptual PRP bottleneck comes in several forms. Primarily, if the bottleneck were perceptual in nature, observation of cross-modal PRP effects would not be expected. Perceptual processing of information presented to different sensory modalities should not yield interference effects such as those found by McCann and Johnston (1992) using auditory and visual stimuli. In addition, a number of studies have obtained underadditive effects by varying stimulus intensity, a variable assumed to have an effect at perceptual encoding (De Jong, 1993, Pashler, 1994b, Pashler & Johnston, 1989). Consequently, the locus of the PRP bottleneck must be after the encoding stage. Several factors presumed to affect the response selection stage of information processing produce an additive effect with SOA (McCann & Johnston, 1992, Schubert, 1999, Van Selst & Jolicœur, 1997). For example, Van Selst and Jolicœur (1997, Experiment 2) demonstrated additive effects of number of response alternatives with SOA, suggesting that the processing bottleneck is located at, or before, response selection.

Further evidence concerning the locus of the bottleneck comes from data demonstrating the effects of manipulating response selection difficulty. McCann and Johnston (1992) assessed the effects of varying stimulus–response symbolic compatibility and found additive effects with SOA, suggesting a bottleneck at or before response selection.

Thus, it appears from the empirical evidence that the PRP bottleneck is most likely located at a central stage of processing, possibly at a stage at which responses are selected. In the present work, we used what is known about the locus of the PRP bottleneck to shed light on the locus of the AB effect by manipulating the same variable (signal probability) in both paradigms and by using locus-of-slack logic to relate the two sets of results.

Signal Probability

In the experiments reported here, a common variable, signal probability, was manipulated both in AB and in PRP experiments. Our strategy was to use a variable that would modulate the AB

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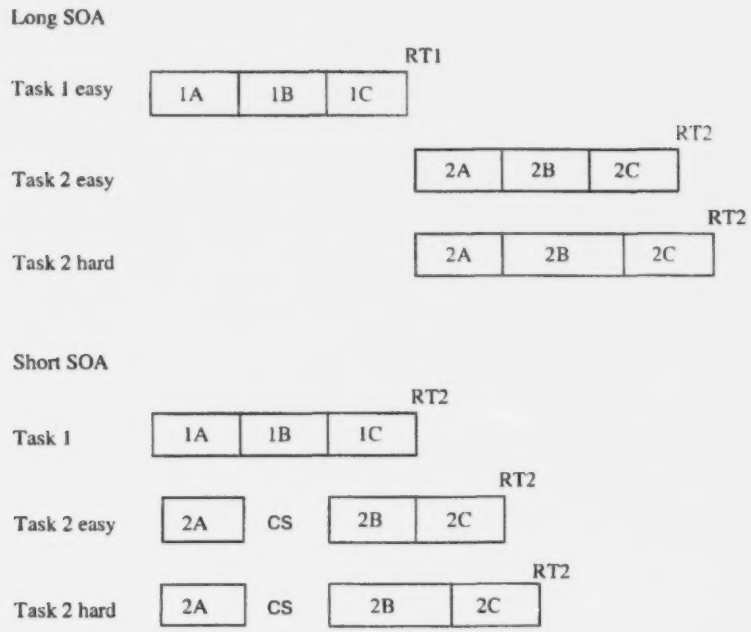

Figure 1 Schematic representation of the effects of manipulating a Task 2 variable that has a locus at or after the psychological refractory period (PRP) bottleneck. Stages 1A–1C reflect the processing necessary for Task 1 with a Task 1 response (RT_1). Stages 2A–2C are processing stages associated with Task 2. In this example, the PRP bottleneck has its locus at Stage B (1B and 2B), and the duration of Stage 2B is affected by the Task 2 manipulation. The locus of cognitive slack (CS) is evident between Stages 2A and 2B at the short stimulus onset asynchrony (SOA), and the effect of varying the duration of a stage of processing at or before the bottleneck will be reflected in mean Task 2 response time (RT_2).

effect and whose effects could be studied in the context of the PRP paradigm. We determined first where signal probability has its effects in relation to the PRP bottleneck, by using locus of cognitive slack logic, and later we established where it has its effects in relation to the AB bottleneck. Assuming that the locus of probability effects does not change across paradigms (an issue that we discuss in more detail in Experiment 3), we could then use the combined results to relate the locus of the AB bottleneck to the locus of the PRP bottleneck.

Two previous experiments suggest that signal probability has an effect that occurs either at or after the PRP bottleneck. In these studies, the effects of varying the probability of the second target stimulus were additive with SOA (Luck, 1998; Van Selst & Jolicœur, 1997). According to the locus of cognitive slack logic, additive effects of relative signal probability and SOA are consistent with the hypothesis that the effect of probability is taking place at, or after, the bottleneck in the PRP paradigm. Signal probability effects have not been studied extensively in the AB paradigm (but see Ward, Downing, Koumos, & Treisman, 1996). The goal of our work was to investigate such effects in more detail and to use them to constrain the locus (or loci) of AB interference. Although these previous results provide some evidence concerning the locus of probability effects relative to the PRP bottleneck, the stimuli, tasks, and probability levels used did not match exactly the conditions that we intended to use in our companion SAB and AB experiments. Given that we intended to compare results across paradigms, it was important to create experimental tasks that were as similar as possible across paradigms.

Studies manipulating signal probability typically show that stimuli presented more frequently lead to faster responses than those presented less frequently (Bertelson & Tisseyre, 1966; LaBerge & Tweedy, 1964; Miller & Hardzinski, 1981; Theios & Walter, 1974). Various views have been proposed as to the fundamental mechanism underlying this finding. Some researchers point to the influence of perceptual factors (Bertelson & Tisseyre, 1966), whereas others attribute the effect to response bias (Sanders, 1970).

Using Sternberg's (1967, 1969) additive factors logic, Miller and Pachella (1973, 1976) observed an interaction of stimulus quality and signal probability in a memory scanning choice RT experiment, suggesting that the effect of probability was occurring at a stage of processing common to the locus of the stimulus quality effect. Given that stimulus quality (contrast) is typically found to produce an underadditive interaction with decreasing SOA (e.g., Pashler & Johnston, 1989), consistent with an early locus of effect, it is not clear why Luck (1998) and Van Selst and Jolicœur (1997) found additive effects of probability and SOA if probability affects an early locus. This apparent discrepancy

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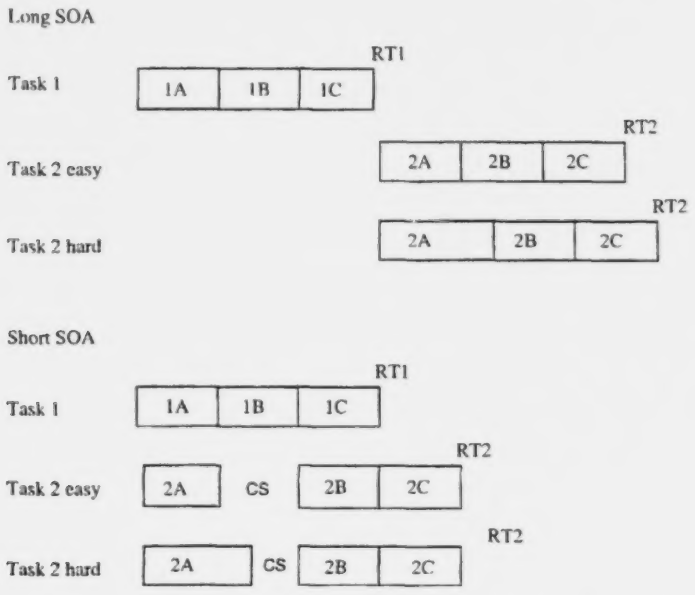

Figure 2 Schematic representation of the effects of manipulating a Task 2 variable that has a locus before the psychological refractory period (PRP) bottleneck. Stages 1A–1C reflect the processing necessary for Task 1 with a Task 1 response (RT_1). Stages 2A–2C are processing stages associated with Task 2. The PRP bottleneck has its locus at Stage B (1B and 2B), and the duration of Stage 2A is affected by the Task 2 manipulation. At a short stimulus onset asynchrony (SOA), the effect of varying the duration of a stage of processing before the bottleneck will be absorbed in the period of cognitive slack (CS) and will not be seen in Task 2 response time (RT_2).

prompted us to perform Experiments 1–3 and to reexamine the relationship between results from the literature on signal probability effects and those from the PRP paradigm, which we summarize after Experiment 3.

The Present Experiments

In Experiments 1–3, we used the PRP paradigm and the locus of cognitive slack logic (Pashler, 1984) to determine where stimulus probability effects take place in relation to the PRP bottleneck. Given the evidence for a high degree of stimulus specificity in effects of signal probability (Miller, 1979; Miller & Hardzinski, 1981), it was important to perform PRP experiments that would produce results directly relevant for the companion AB experiments. Our methods were designed to allow us to compare the results from these experiments directly with those from two additional experiments involving the AB paradigm (Experiment 5) and the SAB paradigm (Experiment 4), a close variant of the standard AB paradigm. The experiments all involved the same stimuli, the same probability manipulation, and, when possible, the same task.

In Experiments 1–3, the PRP experiments, we manipulated signal probability in Task 2. If signal probability affects a late stage of processing at or beyond the PRP bottleneck, then we would expect to observe additive effects of probability and SOA. And, if we suppose that signal probability does have an effect that occurs primarily at the bottleneck stage, then manipulating signal probability in Task 1 of a PRP experiment would be expected to also affect Task 2 (at short SOAs). That is, probability effects would be expected to carry forward from Task 1 to Task 2. This result could arise, however, if probability has an early locus (before the bottleneck), because effects at both prebottleneck and bottleneck stages of Task 1 are predicted to carry forward to Task 2. Consequently, a Task 1 manipulation of signal probability cannot distinguish between a prebottleneck effect and an effect occurring at the bottleneck, whereas a Task 2 manipulation showing additivity with SOA is sufficient to establish a late locus (at or after the bottleneck). It is for these reasons that signal probability was manipulated in Task 2 of the PRP experiments (Experiments 1–3).

Experiment 5 was an AB experiment in which the probability of the first target was manipulated. Experiment 4 was a close variant, what we call an SAB experiment, in which an immediate rather than a delayed response was required in the first task. These experiments were designed to test the carry-forward hypothesis in the context of the AB and SAB paradigms. The task, critical stimuli, and probability manipulation composing Task 1 of Experiment 4 were the same as in Task 2 of the three PRP experiments, allowing direct comparisons of the probability effects across paradigms (see Experiment 3 for further considerations concerning visual masking of the critical stimuli). Indeed, probability effects did carry forward. Finally, in Experiment 5, we removed the

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requirement to make a speeded response in Task 1, thus changing the SAB experiment into an AB experiment, to determine whether the carry forward of the probability effect from Task 1 to Task 2 was due to the speeded response. Carry forward was observed, showing that the effect can be found in both the standard AB paradigm and the SAB paradigm and, thus, that carry forward was not caused by any unique features of the speeded response requirements of the SAB paradigm. The implications of these results for the locus of the bottleneck in the AB and SAB paradigms are discussed in the General Discussion section.

Experiment 1

The goal of Experiment 1 was to constrain the locus of the effect of relative stimulus probability. Applying the locus of cognitive slack logic allowed us to determine the locus of the effect in relation to the locus of the PRP bottleneck. According to this logic, additive effects of probability and SOA indicate that the effect is occurring at or beyond the PRP bottleneck, whereas underadditivity indicates a locus at a stage of processing before the PRP bottleneck.

Method

Participants. Twenty-four undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All of the participants had normal or corrected-to-normal vision. The experiment was run on a super video graphics array (SVGA) color computer screen controlled by a 384, 486, or 586 CPU, programmed with MEL (Schneider, 1988).

Stimuli. Stimuli were auditory tones and uppercase letters. The tones were 400 Hz, 1000 Hz, and 2500 Hz in frequency and were presented through the internal speaker of the computer. The letters were *H*, *O*, and *S*, approximately 0.8° × 1°, presented in the center of the screen in white on a black background.

Procedure. Each trial began with the presentation of one of the tones (Stimulus 1 [S_1]) for a duration of 100 ms, followed by the visual presentation of one of the letters (Stimulus 2 [S_2]). The letter remained on the screen until a response had been made. The SOA between the tone and the letter was varied and could be 50 ms, 200 ms, 450 ms, or 800 ms. A probability variable was associated with the letter stimulus. The relative probability of each letter was 1:4:9, meaning that within every 14 trials one letter (e.g., *H*) was presented 9 times, another letter (e.g., *O*) was presented 4 times, and the remaining letter (e.g., *S*) was presented once. The probability level assigned to each letter was counterbalanced across participants so that, for every 6 participants, all combinations of letters and probability levels were used once. Each complete crossing of SOA, letter (1:4:9), and tones was repeated 4 times, resulting in a total of 672 trials divided equally among four blocks. The experimental trials were preceded by a single block of 48 practice trials.

At the beginning of the experimental session, participants read instructions regarding the procedure and subsequently received verbal instructions. The first task (Task 1) was to identify the tone (low, medium, or high), and the second task (Task 2) was to identify the letter (*H*, *O*, or *S*). RTs to both stimuli were recorded, and responses to the tone and the letter were made by pressing assigned keys on a North American QWERTY keyboard. The assigned keys for the tones were as follows: *Z* for a low tone (400 Hz), *X* for a medium tone (1000 Hz), and *C* for a high tone (2500 Hz). In terms of the letters, these keys were .< for *H*, > for *O*, and /? for *S*. The left hand was used to respond to the tone, and the right hand was used to respond to the letter. After each trial (except the last trial in each block), feedback was given, in the form of adjacent plus or minus signs, as to whether Task 1 and Task 2 had been responded to correctly or incorrectly. The sign on the left referred to Task 1, and the sign on the right referred to Task 2. These feedback symbols also served as a fixation point for the upcoming trial. Each trial was initiated by pressing the space bar, which caused the fixation symbols to disappear and the trial to begin 750 ms later.

Results

Only trials on which responses to Task 1 and Task 2 were correct were used in the analyses. RTs were screened for outliers in each cell for each participant through a modified version of the procedure described by Van Selst and Jolicœur (1994). 2.7% of the trials were rejected on the basis of Task 1 RTs and 1.8% on the basis of Task 2 RTs.

Analysis of Task 1 RTs and accuracy. Cell means for Task 1 RTs were calculated and entered into an analysis of variance (ANOVA) with signal probability (1:4:9) and SOA (50, 200, 450, or 800 ms) as variables. Significant main effects of probability, $F(2, 46) = 3.7$, $p < .034$, $MSE = 6{,}980.54$, and SOA, $F(3, 69) = 3.4$, $p < .023$, $MSE = 3{,}921.47$, were evident. As can be seen in Figure 3 (dashed lines), the effect of probability was strongest at the shortest SOA. Also, at the shortest SOA and at all levels of probability except the Probability 9 condition, mean RT to S_1 was longer than at any other SOA. In general, RT for Task 1 (RT_1) increased as SOA increased from 200 ms to 800 ms, except for the Probability 1 condition, in which RT_1 was faster at the 200-ms and 450-ms SOAs than at the 800-ms SOA. The effects of probability

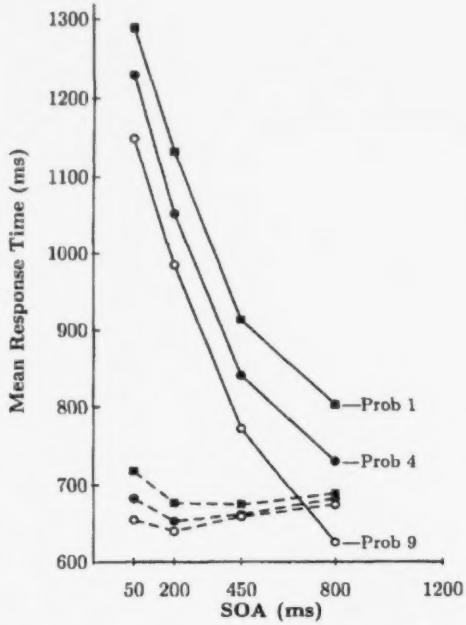

Figure 3. Experiment 1. Mean response times (in milliseconds) for Task 1 (dashed lines) and Task 2 (solid lines) as a function of stimulus onset asynchrony (SOA). The effects of probability (Prob. 1:4:9) of Stimulus 2 as a function of SOA are shown.

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and SOA in Task 1 were eliminated when the 50-ms SOA was not included in the analysis.

Error rates indicated a main effect of SOA, $F(3, 69) = 9.0$, $p < .0001$, $MSE = 0.002$, but no effect of probability, $F(2, 46) = 2.8$, $p > .073$, $MSE = 0.003$, and no interaction, $F(6, 138) = 0.5$, $p > .775$, $MSE = 0.002$. Error rates were slightly higher at the shorter SOAs (50 ms and 200 ms) than at the longer SOAs (450 ms and 800 ms).

Analysis of Task 2 RTs and accuracy. A similar analysis was performed on mean RTs for Task 2 (RT_2s). Figure 3 (solid lines) shows the mean RT for each SOA and each probability level. A significant effect of signal probability was revealed, $F(2, 46) = 28.0$, $p < .0001$, $MSE = 19{,}776.99$. Participants were slowest to respond to the letter that was least likely to appear (Probability 1, $M = 1{,}034$ ms) and fastest to respond to the letter that was most likely to appear (Probability 9, $M = 883$ ms). The mean RT for the Probability 4 condition was intermediate (963 ms). An effect of SOA was also evident: RTs decreased as SOA increased, providing clear evidence of a PRP effect, $F(3, 69) = 455.0$, $p < .0001$, $MSE = 7{,}863.21$. The effect of probability was additive with SOA, with no hint of an interaction, $F(6, 138) = 0.5$, $p > .785$, $MSE = 5{,}333.629$. Although there was a slight fanning out of the means at the longest SOA, this effect was not statistically significant. To ensure that the presence of an interaction was not being washed out by the inclusion of the intermediate SOAs, the data from the 200- and 450-ms SOAs were omitted from the analysis. This mean analysis revealed that the effects of SOA and probability were indeed additive even when only the extreme SOAs (50 and 800 ms) were entered into the analysis, $F(2, 46) = 0.5$, $p < .583$, $MSE = 7{,}802.971$ [1]

Error proportions in Task 2 were highest for the Probability 1 condition (.116) and lowest for the Probability 9 condition (.041), with the Probability 4 condition intermediate between the two (.103), $F(2, 46) = 18.1$, $p < .0001$, $MSE = 0.008$. No effect of SOA was evident, $F(3, 69) = 1.1$, $p > .37$, $MSE = 0.003$, and there was no interaction between SOA and probability, $F(36, 138) = 0.9$, $p > .536$, $MSE = 0.003$.

Discussion

In this PRP experiment, performance on Task 2 was significantly affected by the temporal proximity of the stimuli (S_1 and S_2) associated with the two tasks. As SOA decreased, mean RT in the second task increased. This pattern of results clearly demonstrates a classic PRP effect (Pashler, 1994a). According to the postponement account of the PRP effect, interference in processing the second of two stimuli results from the inability of the processing system to accommodate concurrent processing when processing is common to more than one task. In Experiment 1, processing of the first task, the auditory tone, interfered with the second task of identifying a letter and resulted in a slowing of RT to the letter as SOA decreased.

In addition to the effect of SOA, the results showed an effect of signal probability. RTs were longer for Task 2 letters presented less frequently than for letters presented more frequently. Most important, the effects of SOA and probability were additive. As SOA decreased, RT_2 increased by about the same amount across all probability levels. According to the locus of cognitive slack logic, an additive pattern such as this indicates that the variable—in this case, stimulus probability—is having an effect at or beyond the PRP bottleneck. If the effect of stimulus probability was occurring before the bottleneck stage, Task 2 RTs would have produced an underadditive interaction between probability and SOA as SOA decreased, because the increase in the duration of a processing stage before the bottleneck would have been absorbed in the period of cognitive slack.

The results from Experiment 1 indicate a locus of the probability effect either at or later than the locus of the PRP bottleneck. Evidence in the PRP literature suggests that the PRP bottleneck is located at a late stage of information processing, possibly response selection, or a stage immediately before response selection (McCann & Johnston, 1992; Schubert, 1999; Van Selst & Jolicœur, 1997); thus, the present results lead to the inference that a locus of stimulus probability is at least as late as the locus of the PRP effect, perhaps at the stage of response selection.

Under a simple postponement theory, it is usually assumed that performance on Task 1 would be unaffected by the temporal proximity of S_1 and S_2 (Pashler, 1994a). Contrary to this supposition, however, SOA affected RT to the auditory signal (S_1) in Experiment 1. The general pattern of RT_1 slowing with increasing SOA could possibly result from participants grouping S_1 and S_2 responses on some trials rather than responding to the first stimulus independent of the second stimulus. This strategy would entail saving the first response until the second response has been selected and would result in a slowing of RT_1 correlated with the time between onset of the two stimuli, increasing as SOA increases (Pashler, 1984).

The effects on RT_1 in this experiment were small and not unusual for PRP data. Possible explanations might exist (e.g., grouping, see Pashler & Johnston, 1989), but, at present, the cause of the specific effects of Task 2 manipulations on Task 1 performance in Experiment 1 is unclear. The results of interest are those from Task 2, and, although effects on Task 1 were found in the data, these effects were much smaller than those in Task 2, as demanded by a postponement account. Thus, it appears reasonably safe to focus the analysis on the results from Task 2.

However, because of the influence of SOA on RT_1 in the present experiment, it seems appropriate to discuss a possible capacity-sharing account of the results. Tombu and Jolicœur (2001) derived the properties of a class of models in which early and late stages of processing are capacity free but one or more central stages share limited processing capacity. These models are similar to all-or-none bottleneck models (illustrated in Figures 1 and 2) in that prebottleneck and postbottleneck stages are capacity free but differ from them in that the central stages can share the available capacity in a graded fashion rather than capacity being allocated to each task in an all-or-none fashion. In fact, all-or-none bottleneck models represent a special case of the central capacity-sharing model in which the sharing proportion (the parameter in the model that controls how much capacity is allocated to each task) is set to

[1] To ensure that the effects of probability and SOA in Task 1 were not biasing our interpretation of these effects in Task 2, we conducted an analysis of covariance, with Task 1 mean RT as the covariate. This analysis revealed that the interaction between probability and SOA remained nonsignificant when RT_1 was covaried out, $F(6, 138) = 1.5$, $p > .20$, $MSE = 7.117.0$. We thank Tom Carr for suggesting this procedure.

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1.0 for one of the tasks, leaving a capacity of zero for the other task. Tombu and Jolicœur (2001) showed that this class of models makes predictions similar to those of all-or-none bottlenecks, but with a number of notable differences. In particular, the model predicts that RT_1 will increase as SOA is reduced when sharing takes place. Most important, however, as mentioned earlier, the model predicts additivity of SOA and bottleneck or postbottleneck Task 2 effects, underadditivity of prebottleneck Task 2 effects, and carry forward of bottleneck or prebottleneck Task 1 effects, as for all-or-none models. Consequently, we can interpret these effects and arrive at correct conclusions regardless of whether central capacity is allocated in a graded fashion or in an all-or-none fashion.

In the present experiment, the effects of SOA on Task 1 were not entirely consistent with either model, because Task 1 RTs tended to increase as SOA increased at all but the shortest SOA (50 ms). The observation that RT_1 increased with increasing SOA does not support a capacity-sharing account of the results. It should be noted that the percentage of errors on Task 1 decreased as RT slowed, suggesting the possibility of a speed–accuracy trade-off. As participants responded more quickly in Task 1, they in turn committed more errors. In Task 2, there was no effect of SOA on errors, indicating that there was no such trade-off.

Experiment 2

Although the results of Experiment 1 provided no statistical indication of an interaction between probability and SOA ($F < 1$), Task 2 performance at the longest SOA (800 ms) in the Probability 9 condition appeared to deviate slightly from strict additivity. There was a larger RT_2 difference between each probability level at this SOA than at other SOAs. We conducted Experiment 2 to be certain that this pattern was not indicative of weak underadditivity that we might have failed to detect. One explanation for the slight deviation from additivity at the longest SOA is the possibility that this data point represents a slight response bias. Participants may have expected—and prepared the response for—the most frequently presented stimulus, which could have introduced a response bias. Such a response bias might take time to develop after the processing required to perform Task 1, and thus it might be strongest at the longest SOA. In light of this possibility, it would be of interest to observe the data pattern under conditions in which the longest SOA is more than 800 ms. For this reason, an SOA of 1,200 ms was included in Experiment 2. Another reason to include the 1,200-ms condition is to ensure a proper baseline condition in which RTs in the second task have reached asymptote. In addition, we decided to add a large percentage of catch trials (33%) in which no S_2 was presented. We hoped that this modification to the design would discourage participants from expecting the most likely stimulus and that they would wait for S_2 and process it in an unbiased way. The addition of S_2-absent trials also served to equate the PRP experiments with the AB and SAB experimental design in which T_1-absent trials were included as control trials (see Experiments 4 and 5).

Method

The method and procedure for Experiment 2 were similar to those of Experiment 1.

Participants. Twenty-four undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All of the participants had normal or corrected-to-normal vision.

Stimuli. Stimuli for Task 1 were identical to those used in Experiment 1.

Procedure. Modifications to the design were as follows. In addition to the SOAs used in Experiment 1, a 1,200-ms condition was included. Also, a condition in which there was no S_2 letter was added. On two thirds of the trials, an S_2 letter (*H*, *O*, or *S*) was presented (S_2-present trials). The remaining trials were S_2-absent trials. The relative probability (1:4:9) associated with each letter was counterbalanced, as before, between participants. On the S_2-present trials, participants responded using the appropriate keys, which were the same as those assigned in Experiment 1. On S_2-absent trials, an audible chirp sound was presented at the 1,600-ms SOA to inform individuals that no letter would appear and that they should press the space bar to continue to the next trial. The chirp sound was created by presenting a sequence of four 50-ms tones (632 Hz, 1581 Hz, 632 Hz, and 1581 Hz). RTs for Task 1 were recorded on every trial, and RTs for Task 2 were recorded on the S_2-present trials.

Because of the addition of the S_2-absent condition and the 1,200-ms SOA condition, the number of trials increased, from 672 in Experiment 1 to 1,260 in the present experiment. As a result of the length of this experiment, the trials were divided into two sessions conducted over 2 days. In the first session, participants completed a practice block of 100 trials. This practice session was followed by four blocks of experimental trials, with each block consisting of 105 trials. In the second session, on Day 2, the practice block was reduced to 40 trials and was followed by eight blocks of experimental trials, 105 trials in each, resulting in a total of 1,260 experimental trials over the course of the two sessions.

The procedure was identical to Experiment 1 except that participants were informed that on some trials the second stimulus (letter) would not be present and in those cases they would hear a chirp sound, at which time they were to press the space bar. As a means of minimizing the possibility that participants would wait for the sound rather than preparing for a possible response, which might be particularly likely at the longest SOA (1,200 ms), they were instructed to be prepared to respond to a letter right up until they heard the chirp sound. Note that the most frequent type of trial in this experiment was the S_2-absent trial (33% of trials). If participants were to bias their response in any way, they would probably show a bias for using the space bar rather than a response key for one of the critical S_2 target letters.

Results

RT outliers for correct responses on both tasks were removed from the data set in a similar fashion to Experiment 1. This led to rejection of 2.4% of the trials on the basis of Task 1 RTs and 1.6% of the trials on the basis of Task 2 RTs.

Analysis of Task 1 RTs and accuracy. Cell means for Task 1 RTs were calculated and entered into an ANOVA with signal probability in Task 2 (1:4:9) and SOA (50, 200, 450, 800, or 1,200 ms) as variables. Significantly slower RTs at the shorter SOAs (50 ms and 200 ms) were responsible for the effect of SOA in Task 1, $F(4, 92) = 31.8$, $p < .0001$, $MSE = 4{,}418\,873$. This pattern can be seen in Figure 4 (dashed lines). There was no effect of signal probability, $F(2, 46) = 1.1$, $p > .33$, $MSE = 3{,}729.968$, and no interaction between probability and SOA, $F(8, 184) = 1.3$, $p > .247$, $MSE = 3{,}342.201$. Furthermore, no main effects in the error data were evident, and there was no interaction between SOA and probability, $F(8, 184) = 0.4$, $p > .917$, $MSE = 0.003$. The overall accuracy rate on S_2-absent trials was .99 (proportion correct), and the mean RT for S_1 when S_2 was absent was 590 ms.

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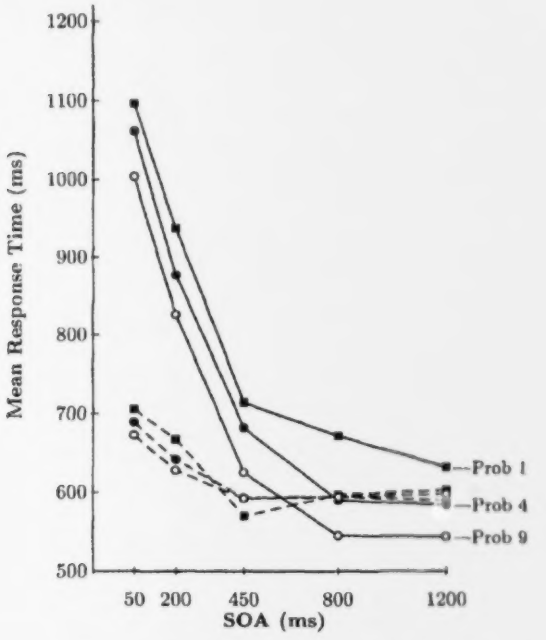

Figure 4 Experiment 2: Mean response times (in milliseconds) for Task 1 (dashed lines) and Task 2 S_2-present trials (solid lines) as a function of stimulus onset asynchrony (SOA). The effects of probability (Prob. 1 4 9) of S_2 as a function of SOA are shown. S_2 = Stimulus 2.

Analysis of Task 2 RTs and accuracy. Mean Task 2 RTs are shown in Figure 4 (solid lines). Significant effects of probability, $F(2, 46) = 34.2$, $p < .0001$, $MSE = 9{,}004.972$, and SOA, $F(4, 92) = 252.1$, $p < .0001$, $MSE = 11{,}619.403$, were found, with no evidence of an interaction between these variables, $F(8, 184) = 0.8$, $p > .582$, $MSE = 3{,}618.979$. RTs to the letter presented most frequently (Probability 9) were significantly faster ($M = 709$ ms) than RTs to the lowest-probability letter ($M = 810$ ms). Mean RT in the Probability 4 condition was intermediate between the Probability 1 and 9 conditions (759 ms). Overall, RT_2 decreased as SOA increased, being slowest at the shortest SOA and fastest at the longest SOA, although at the 800-ms and 1,200-ms SOAs, RT_2 was almost equivalent across the Probability 4 and 9 conditions.[2]

Only a main effect of probability was evident when mean error rates were analyzed, $F(2, 46) = 11.1$, $p < .001$, $MSE = 0.012$. Participants were most accurate on trials that made up the Probability 9 condition (error rate: .036), followed by the Probability 4 condition (error rate: .077), they were least accurate in the Probability 1 condition (error rate: .105).

Discussion

In this experiment, stimulus probability produced effects similar to those found in Experiment 1. Mean RT in Task 2 was shortest for the probability condition in which the S_2 letter was presented most frequently and longest in the condition in which the letter was presented least frequently. Most important, the effects of stimulus probability were additive with SOA, showing that probability was having an effect at a stage of processing at or beyond the PRP bottleneck.

The 1,200-ms-SOA condition was added in this experiment to ascertain whether or not participants reached asymptote in their performance on Task 2 at the 800-ms SOA. In the Probability 4 and 9 conditions, RTs were no different when the SOA was 1,200 ms than when it was 800 ms. The absence of any hint of a statistical interaction between probability and SOA suggests that the deviation from strict additivity in Experiment 1 was probably due to measurement error. The relatively flat RT_2s across the 800- and 1,200-ms-SOA conditions in Experiment 2 suggest that performance was near asymptote levels in the 800-ms condition. Thus, in Experiments 1 and 2, the absence of a significant interaction between probability and SOA could not have been due to a failure to test performance at an SOA producing a near asymptote RT_2, as required by locus-of-slack logic. The results of Experiment 2 converge nicely with those of Experiment 1 in showing that the effects of the probability of S_2 are additive with SOA under the conditions tested in these experiments. Thus, the results show that probability effects in these experiments occurred at or later than the locus of the PRP bottleneck.

Also, in Experiment 2, an effect of SOA on Task 1 RTs was present. This effect was entirely due to the contribution of the two shortest SOAs (50 and 200 ms). There was no difference in RT_1 at longer SOAs (450, 800, and 1,200 ms), but at the shorter SOAs (50 and 200 ms) RT_1 increased as SOA decreased. As discussed in Experiment 1, this pattern of results indicates the possibility of graded capacity sharing at one or more central stages of processing.

Experiment 3

Experiments 1 and 2 determined that the effect of relative signal probability is occurring at, or after, the locus of the PRP bottleneck. Our goal, however, was to go beyond these experiments and to use this finding to constrain the locus of AB effects, by first determining the locus of stimulus probability in relation to the AB and then ascertaining the relationship between the loci of the AB and PRP phenomena. The rationale behind this analysis rests on the assumption that the locus of stimulus probability is invariant, regardless of whether we are dealing with a PRP or an AB paradigm. Therefore, a concern that must be addressed is the possibility that the locus of probability is not the same in the PRP and AB paradigms.[3] The fact that findings in the literature are inconclusive in resolving a locus for the probability effect may in itself be evidence for the existence of more than one locus.

The AB and PRP paradigms differ in ways that could jeopardize the assumption that manipulations in one paradigm affect the same processing locus in the other paradigm. In particular, the rapid stimulus presentation integral to the AB paradigm places large

[2] As in Experiment 1, an analysis of covariance was conducted with RT_1 as the covariate. This analysis was performed to ensure that the effect of SOA in Task 1 was not influencing the same effect in Task 2. The analysis revealed that the interaction of probability with SOA in Task 2 remained nonsignificant when RT_1 was covaried out, $F(8, 138) = 0.8$, $p > .60$, $MSE = 3{,}823.98$.

[3] We thank James Johnston for this insight.

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demands on stimulus perception and encoding. In contrast, the PRP paradigm may place fewer demands on early perceptual encoding and more on the later stage of response selection (although this latter possibility does not apply to the first task of the SAB paradigm, which is speeded and requires the immediate selection of a response). If probability affects different stages of processing, it could have a larger effect at perceptual encoding in the AB paradigm and a larger effect at response selection in the PRP paradigm.

One way to determine whether or not a locus of probability exists at an early stage of processing would be to modify the PRP paradigm so that it is more perceptually demanding and more similar to the viewing conditions in effect in the AB paradigm. We did this in Experiment 3 by presenting the critical stimulus briefly, sandwiched between a premask and a postmask. These viewing conditions were very similar to those of the target stimuli in Experiments 4 and 5. Giesbrecht and Di Lollo (1998) and Brehaut, Enns, and Di Lollo (1999) have shown, in a number of different tests, that inclusion of a single posttarget character has the same masking effects, in the AB paradigm, as an entire stream of trailing characters. If increasing the perceptual difficulty of the PRP task shifts the locus of signal probability effects to an earlier point, then we should now observe an underadditive interaction between probability and decreasing SOA. Finding such an interaction would complicate the empirical picture, to be sure, and this is discussed in detail in the general discussion of Experiments 1–3.

Method

The method and procedure for Experiment 3 were similar to those of Experiment 2 with the exception that participants were given a choice of dividing the experimental sessions over 2 days or taking a 15-min break before continuing on to the next session. Also, S_2 was masked (as described subsequently).

Participants. Twenty-four undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All of the participants had normal or corrected-to-normal vision.

Stimuli. Stimuli for Task 1 and Task 2 were identical to those used in Experiment 2, except that pre-S_2 and post-S_2 masks were added and the duration of S_2 was set to 100 ms. Each mask was a randomly chosen uppercase letter presented for a duration of 100 ms with no interstimulus interval. The premasks and postmasks were never the same letter, and those letters used as S_2 targets (i.e., *H*, *O*, and *S*) were excluded from the set.

Results

RT outliers for correct responses on both tasks were removed from the data set in a similar fashion to Experiments 1 and 2. This led to rejection of 1.77% of the trials on the basis of Task 1 RTs and 2.61% of the trials on the basis of Task 2 RTs. Two participants were eliminated from the data set as a result of their low accuracy rates on Task 1 and Task 2.

Analysis of Task 1 RTs and accuracy. Cell means for Task 1 RTs were calculated and entered into an ANOVA with signal probability of Task 2 (1-4-9) and SOA (50, 200, 450, 800, or 1,200 ms) as variables. As in Experiment 2, significantly longer RTs at the shorter SOAs (50 ms and 200 ms) produced a main effect of SOA in Task 1, $F(4, 92) = 24.4$, $p < .0001$, $MSE = 7{,}947.963$. This pattern can be seen in Figure 5 (dashed lines). Although there was no effect of signal probability, $F(2, 46) = 0.1$, $p > .93$, $MSE = 1{,}898.801$, the interaction between probability and SOA was significant, $F(8, 184) = 2.2$, $p < .031$, $MSE = 2{,}814.479$.

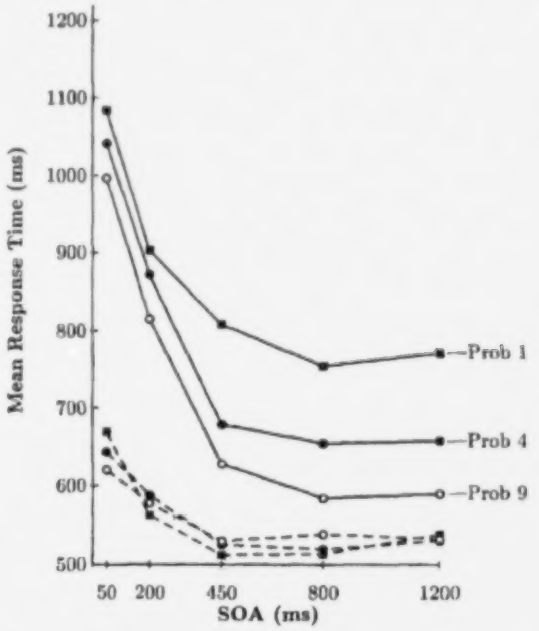

Figure 5. Experiment 3. Mean response times (in milliseconds) for Task 1 (dashed lines) and Task 2 S_2-present trials (solid lines) as a function of stimulus onset asynchrony (SOA). The effects of probability (Prob. 1 4 9) of S_2 as a function of SOA are shown. S_2 = Stimulus 2.

With respect to the error data for Task 1, only the effect of SOA was significant, $F(4, 92) = 5.0$, $p < .01$, $MSE = 0.004$. In general, accuracy increased as SOA increased. There was no effect of probability, $F(2, 46) = 0.3$, $p > .76$, $MSE = 0.001$, but the two-way interaction between SOA and probability approached significance, $F(8, 184) = 1.9$, $p < .06$, $MSE = 0.003$. The overall accuracy rate in Task 1 on S_2-absent trials was .94 (proportion correct), and mean RT was 535 ms.

Analysis of Task 2 RTs and accuracy. A similar analysis, shown in Figure 5 (solid lines), was performed on mean RTs for Task 2. Significant effects of probability, $F(2, 46) = 25.5$, $p < .0001$, $MSE = 23{,}845.73$, and SOA, $F(4, 92) = 176.8$, $p < .0001$, $MSE = 10{,}641.03$, were found, as well as an interaction between these variables, $F(8, 184) = 3.8$, $p < .001$, $MSE = 4{,}817.55$. RT to the letter presented most frequently (Probability 9) was significantly faster ($M = 723$ ms) than that to the lowest probability letter ($M = 864$ ms). Mean RT in the Probability 4 condition was intermediate between the Probability 1 and 9 conditions (781 ms). Overall, RT_2 decreased as SOA increased, being slowest at the shortest SOA and fastest at the longest SOA, although RT_2 was almost equivalent across all probability levels at the 800- and 1,200-ms SOAs.

A closer examination of the RT_2 results suggested that (a) the interaction between SOA and probability was caused by a pattern of means that was different for the Probability 1 condition than for

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the two higher probability conditions and (b) the Probability 4 and 9 conditions were actually additive rather than underadditive with SOA, as the omnibus interaction suggested. Indeed, additional analyses revealed that the Probability 4 and Probability 9 conditions were additive with SOA, $F(4, 92) = 1.0$, $p > .43$, $MSE = 1{,}587.75$, whereas the comparison of Probabilities 1 and 4 was underadditive with SOA, $F(4, 92) = 3.3$, $p < .015$, $MSE = 7{,}070.53$. Consequently, as expected, the interaction of SOA with probability was mainly due to a difference between the lower probability condition and the other two. In the error data, main effects of SOA, $F(4, 92) = 9.5$, $p < .0001$, $MSE = 0.008$, and probability, $F(2, 46) = 10.3$, $p < .001$, $MSE = 0.046$, were evident, with no interaction between the two, $F(8, 184) = 0.9$, $p > .55$, $MSE = 0.009$.

Experiments 2 and 3 differed mainly in the viewing conditions of S_2 (unmasked in Experiment 2 vs. masked in Experiment 3). This manipulation allowed us to examine the effects of pattern masking in the context of the PRP paradigm. Figure 6 shows mean RTs in Task 2 depending on whether S_2 was masked or not. The effects of masking were clearly underadditive with decreasing SOA. Masking slowed responses to S_2 at long SOAs but had no effect at short SOAs, $F(4, 184) = 6.7$, $p < .0001$ (test for the interaction between experiment and SOA in a separate ANOVA). These results show that, under the present conditions, masking exerted most of its effect by lengthening the duration of an early stage of processing.

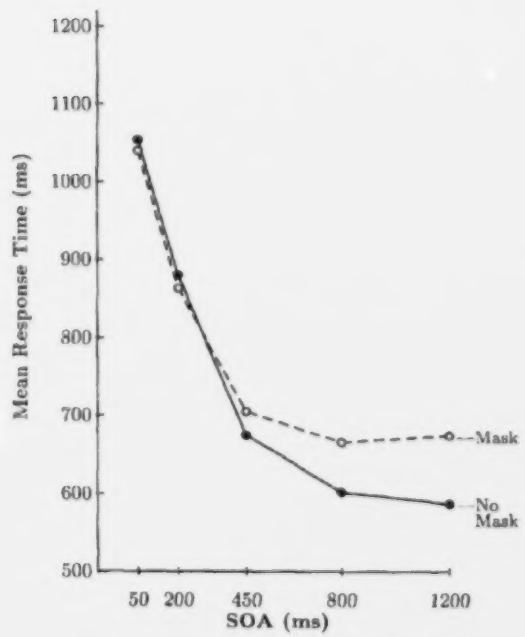

Figure 6 Experiments 2 and 3. Mean response times (in milliseconds) for Task 2 S_2-present trials as a function of the presence or absence of masking (Experiment 2, no mask, solid lines; Experiment 3, masked, dashed lines) and stimulus onset asynchrony (SOA). Masking slowed responses to S_2 at long SOAs but had no effect at short SOAs. S_2 = Stimulus 2.

Discussion

The results were clear cut. When S_2 was masked, an underadditive interaction between probability and decreasing SOA was observed. These results suggest strongly that, under these viewing conditions, signal probability had some of its effect before the PRP bottleneck. Most important, however, was the finding that effects of probability remained additive with SOA when only moderate to high levels of signal probability were considered. This issue is discussed in more detail in the next section.

Discussion: Experiments 1–3

The results of Experiments 1–3 provide new constraints and insights on the nature of signal probability effects. In Experiments 1 and 2, in which T_2 was not masked, signal probability effects in Task 2 were additive with SOA. Interpretation of these results is straightforward. Under the present testing conditions, the manipulation had its effects either during or after the PRP bottleneck (Pashler & Johnston, 1989). Given what is known about the probable locus of the PRP bottleneck, this suggests a relatively late locus for probability.

In Experiment 3, in which T_2 was masked, signal probability and SOA interacted. Probability effects were larger at longer SOAs than at shorter SOAs. This general pattern of results suggests that, at least some of the time, part of the probability effect occurred before the bottleneck. Additional analyses showed that SOA and probability were additive if the analysis was restricted to the Probability 4 and 9 conditions, whereas each of these conditions produced an interaction with SOA when combined with the Probability 1 condition. Furthermore, there were obvious residual effects of probability at the shortest SOA, suggesting that only a portion of the probability effect was absorbed into slack.

Our interpretation of these additional analyses is that signal probability generally has an effect that occurs at or after the PRP bottleneck, even if T_2 is masked, when signal probability ranges from moderate to high. However, if T_2 is masked and has a relatively low probability, then probability affects an additional locus before the bottleneck. It is this early effect for degraded low-probability targets that is absorbed into slack and produces the observed underadditive interactions with decreasing SOA. This interpretation is illustrated in Figure 7 (top).

If T_2 is presented without masks, as in Experiments 1 and 2, then probability has an effect that is primarily late (at or after the PRP bottleneck). Under conditions of perceptual stress (e.g., T_2 masked) and very low probability levels, as in Experiment 3, probability also affects an early stage. In this view, increasing the perceptual difficulty of the task modifies the effects of probability by inducing an additional and earlier locus of action. However, longer processing at the early locus occurs only for low-probability signals. The bottom panel of Figure 7 shows the loci of probability predicted when perceptual stress is induced by reducing stimulus contrast.

How does this interpretation, which accounts nicely for the results of Experiments 1–3, jibe with the literature on probability effects? To provide a satisfactory test of our model, an experiment

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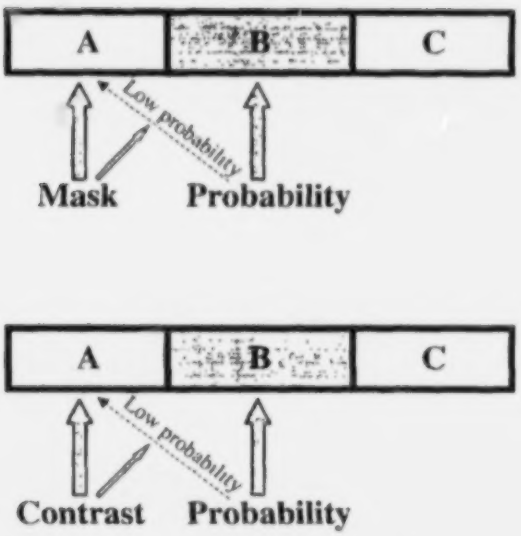

Figure 7 Proposed loci of probability, in relation to the psychological refractory period bottleneck (Stage B), as a function of level of probability (low or moderate) and whether Target 2 is presented under conditions of perceptual stress (top: masked vs. unmasked; bottom: low vs. high contrast).

must include at least three levels of (moderate or low) probability, allowing examination of effects of probability at different points. Also, there should be at least two levels of perceptual difficulty. We found six experiments in the literature that met these requirements, and the results are compiled in Table 1. In these experiments, the contrast of visual stimuli was manipulated in conjunction with a manipulation of stimulus probability. Stimuli were presented one at a time and required a speeded response. As expected, longer RTs tended to be found when probability was lower and when contrast was lower. In addition, in some experiments but not others, an interaction between contrast and probability was found. This interaction is generally interpreted as evidence for an early locus for probability, given that contrast is typically believed to also have an early locus (Sternberg, 1969). To support our proposal, the pattern of interaction between contrast and probability would have to exhibit the following form: (a) There should be essentially additive effects of contrast and probability for moderate to high signal probability levels, (b) interactions between contrast and probability should occur only for low probability levels, and (c) these interactions should tend to occur only under conditions of increased perceptual difficulty.

In Figure 8, we plot the RT difference between the low-contrast and high-contrast conditions, at each of the four probability levels, for the six data sets compiled in Table 1. For three of the data sets (C, D, and E), the effects of contrast were roughly constant across all probability levels, that is, contrast and probability had additive effects over the entire range of probability values. For the other three (A, B, and F), there was an interaction between contrast and probability: Larger contrast effects were found for lower probability levels.

Two aspects of these results provide good support for our presentation. First, even when contrast and probability interacted (when the entire range of probabilities was considered), there was no change in the size of the contrast effect across the two highest probability levels. That is, at these probability levels, contrast and probability always had additive effects. Second, the observed interaction between contrast and probability tended to increase in magnitude as the mean size of the contrast effect increased. We assume that the stimuli in the low-contrast condition became more difficult to perceive as the size of the contrast effect increased. The

Table 1
Mean Response Times (in Milliseconds) in Six Experiments as a Function of Stimulus Contrast and Probability (Estimated From Graphs)

Study and experiment	Stimulus contrast	Probability			
		.025	.075	.125	.275
Miller and Pachella (1973)					
Experiment 1 (manual response)	Normal	580	542	527	508
	Low	872	798	768	746
Experiment 2 (naming response)	Normal	517	509	505	500
	Low	770	728	722	706
Miller and Pachella (1976)					
Experiment 2 (manual response)	Normal	917	812	755	695
	Low	1,020	930	878	800
Experiment 3 (manual response)	Normal	798	725	678	622
	Low	860	780	745	680
Stanovich and Pachella (1977)					
Experiment 1 (manual response)	Normal	815	700	660	610
	Low	1,005	880	830	780
Experiment 1 (naming response)	Normal	535	540	545	540
	Low	800	770	740	740
Mean difference between normal and low stimulus contrast		191	172	170	163

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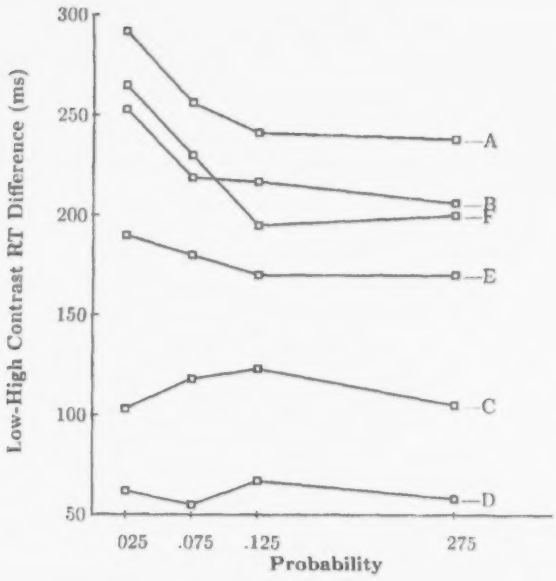

Figure 8 Plot of mean response time (RT, in milliseconds) difference between low- and high-contrast conditions at each of the four probability levels for the six data sets compiled in Table 1 (A = Miller & Pachella, 1973, Experiment 1, B = Miller & Pachella, 1973, Experiment 2, C = Miller & Pachella, 1976, Experiment 2, D = Miller & Pachella, 1976, Experiment 3, E = Stanovich & Pachella, 1977, Experiment 1, manual response, F = Stanovich & Pachella, 1977, Experiment 1, naming response)

fact that the interaction, for low probability levels, increased in magnitude as perceptual difficulty increased is nicely consistent with our model and with the results of Experiments 1–3. Figure 7 (bottom) shows how the results from Table 1 and Figure 8 can be interpreted within our framework. Generally, probability affects a later stage of processing, whereas contrast affects an earlier stage. However, when contrast levels are sufficiently low, the increase in perceptual difficulty causes probability to also have an influence at an earlier locus, but only for low probabilities. The added effect at the earlier locus now causes the interaction between probability and contrast that is confined to lower probability levels. This account of the pattern of results for contrast and probability is structurally isomorphic with our account of the effects of masking and probability in the context of the PRP paradigm.

The results of Experiments 1–3 and a reexamination of the earlier work in which probability and contrast were jointly manipulated suggest that probability can affect two processing loci, one relatively early and one relatively late in the information-processing sequence. The early locus, under appropriate conditions, leads to interactions between contrast and probability, on the one hand, and to interactions between SOA and probability, on the other. However, the results suggest strongly that two conditions are necessary for probability to have an early effect. First, the probability levels must be quite low. Second, the stimuli must be presented under conditions of high perceptual difficulty. Sufficiently difficult perceptual conditions can be produced either by very low contrast levels or by masking with pattern masks. Finally, we want to make it clear that we are not claiming that these conclusions necessarily apply to all manipulations of signal probability for all possible stimuli and tasks. Rather, we intend our conclusions to apply to the kinds of task and stimuli used here and in experiments such as those of Miller and colleagues, namely choice tasks with stimuli such as letters, digits, words, and other similar forms. Whether these conclusions apply to other domains is an interesting question for future research.

Experiment 4

Experiments 4 and 5 were designed as a pair of complementary experiments involving paradigms different from those used in Experiments 1–3. Experiment 4 was an SAB experiment, that is, an AB experiment in which a speeded response was required in Task 1. A typical unspeeded and delayed response was required in Task 2. The only difference between Experiments 4 and 5 was that an immediate and speeded response was required in Task 1 of Experiment 4, whereas a deferred and unspeeded response was required in Experiment 5.

We performed Experiment 4 primarily for two reasons. First, we wished to discover whether a manipulation of signal probability in Task 1 in an SAB paradigm would carry forward and affect performance in Task 2. As discussed in the introduction, according to both all-or-none and central capacity-sharing bottleneck models of dual-task interference, effects occurring at or before the bottleneck in Task 1 are expected to carry forward to Task 2. Therefore, if we observe effects in Task 2 resulting from a manipulation of signal probability in Task 1, we will be able to conclude that probability effects in this paradigm occur either at the bottleneck stage or before.

The second motivation for Experiment 4 was to bridge the gap between the PRP experiments reported in the first portion of the article and Experiment 5, an AB experiment. This was one of the reasons for using a speeded response in Task 1 of Experiment 4. Indeed, this task, which was the one in which signal probability was manipulated, was very similar to Task 2 in Experiments 1–3. The target stimuli were the same (*H*, *O*, or *S*). Furthermore, signal probability was manipulated in the same way. As before, there was a ratio of 1:4:9 in the relative frequencies of the three target letters. The task required the same immediate and speeded response, executed with the same fingers of the same hand and the same response keys on the same keyboard as in the PRP experiments. In other words, the basic task to be performed was very similar across the two paradigms.

Although the tasks were very similar, there were some differences. The stimuli were presented through RSVP in Experiment 4 but not in Experiments 1 and 2. In Experiments 1 and 2, the critical stimulus was presented without masks, and it remained in view until the response (as in the typical PRP paradigm). However, in Experiment 3, the stimulus was presented for the same duration as in Experiment 4 (100 ms), and it was masked in a similar way by presenting it between two other characters at the same location. Thus, the sequence of visual stimuli in Experiment 3 was a short RSVP stream. The main difference between Experiments 3 and 4, in terms of the viewing conditions for the target subject to the signal probability manipulation, was the length of the RSVP

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stream. The stream consisted of only three items in Experiment 3, whereas it was much longer in Experiment 4.

There were other differences between Experiments 3 and 4. A speeded response was required in Task 2 of Experiment 3 but not in Experiment 4, and this is the defining difference making Experiment 3 a PRP experiment and Experiment 4 an SAB experiment, because both involved a speeded first task. Another difference was that signal probability was manipulated in Task 2 of Experiment 3 but in Task 1 of Experiment 4. The reasons for this difference hinge on the logical structure of the argument we wish to make vis-à-vis the locus of signal probability effects in the two paradigms. In the case of the PRP paradigm, the goal was to establish that at least some part of the effect, under experimenter control, could be safely assumed to occur at or after the bottleneck. This required a manipulation in Task 2 (see the discussion of locus-of-slack logic in the introduction). On the other hand, for the SAB and AB paradigms, in which Task 2 was not speeded, locus-of-slack logic could not be used in a straightforward way, because results from Task 2 were based on accuracy rather than RT (there was no RT given that the second task was not speeded). The carry-forward prediction for a bottleneck (or prebottleneck) Task 1 effect to Task 2, however, can be applied safely to accuracy data, because observing the presence or absence of carry forward does not hinge on exact quantitative relationships (unlike measuring the difference between additive and underadditive effects with SOA for speeded Task 2 experiments). This issue is discussed further in the General Discussion section.

It was sufficient, for our purposes, to manipulate probability in Task 1 and to observe whether carry forward was found or not. Accordingly, we manipulated probability in Task 1 of Experiment 4. According to postponement models, the effects of factors at or before the dual-task bottleneck, when manipulated in Task 1, should also be reflected in Task 2. At very short SOAs, the magnitude of the effects in Task 2 should be large, whereas the coupling between Task 1 and Task 2 should weaken as SOA is lengthened. When the time between stimulus onset is short, increasing the duration of a Task 1 stage at or before the bottleneck will result in an increase in RT_1 and an equal delay in the onset of processing necessary for Task 2, assuming that Task 2 requires a mechanism that is busy with Task 1. As SOA increases, processing associated with Task 1 will have begun, or be complete, before the onset of the Task 2 stimulus. Consequently, any delay in Task 2 processing will diminish as SOA increases. These predictions have been verified a number of times in published articles and in numerous unpublished experiments from our laboratory (e.g., Pashler, 1994b; Van Selst et al., 1999). Furthermore, a Task 1 manipulation that has its effect after the dual-task bottleneck should not carry forward to Task 2 (see Pashler, 1994b).

Manipulating probability in Task 2 of an AB experiment could perhaps be interesting in its own right. However, interpretation of the results would not be as straightforward as one might think. It is not clear that the quantitative predictions of the locus-of-slack approach developed for the analysis of Task 2 RTs in the PRP paradigm can simply be applied in the same way to the analysis of Task 2 accuracy scores in the AB paradigm (see Schweickert, 1985). The qualitative prediction that a bottleneck or prebottleneck effect in Task 1 should carry forward to Task 2 performance, however, is sound because it does not depend on precise quantitative relationships. The central interference theory proposed by Jolicœur (1998, 1999a, 1999b, 1999c; Jolicœur, Dell'Acqua, & Crebolder, 2001; Ross & Jolicœur, 1999) and Chun and Potter's (1995) two-stage model predict that a Task 1 manipulation that has an effect at or before the locus of the critical stage causing the AB should also have an effect on Task 2. In these models, processing of T_1 delays the onset of processing of T_2, and longer delays cause larger deficits in Task 2. This can only happen, however, if the stage that is delayed in Task 1 occurs at or before the locus of dual-task interference. Any effects after this point will not carry forward, because these effects do not delay processing in Task 2. All published models of the AB effect predict that if a Task 1 manipulation has its effect after the locus of the AB effect, there should be no effect on Task 2. In Experiments 4 and 5, we used these predictions to examine the locus of signal probability effects relative to the stage or stages of processing causing the dual-task deficit in Task 2 of these experiments.

Method

Participants. Seventy-eight undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All of the participants had normal or corrected-to-normal vision.

Stimuli. Experimental sessions were run on an SVGA color monitor controlled by a 384, 486, or 586 CPU, programmed with MEL (Schneider, 1988). Stimuli were uppercase letters presented in RSVP in the center of the screen for a duration of 100 ms with no interstimulus interval. All of the letters were presented in white on a black background, except the target stimulus (T_1), which was red. Background stream items were selected at random without replacement from the letters of the alphabet excluding *H*, *O*, *S*, *X*, and *Y*. There were 6–9 letters (this number was selected randomly at run time) presented before T_1 and 9–12 letters (also selected randomly at run time) presented after T_1. The probe stimulus (T_2) could occur in any one of the eight positions immediately following T_1 (henceforth referred to as T_1–T_2 lag [1–8]). This procedure ensured that there were always 1–4 letters presented after T_2, thus providing the T_2 masking conditions necessary to observe an AB effect (Giesbrecht & Di Lollo, 1998; Jolicœur, 1999b).

Letters subtended about 1° of visual angle. The white letters had an approximate luminance of 25 cd/m^2 on a black background of less than 1 cd/m^2. Red stimuli had an approximate luminance of 26 cd/m^2. Because the experiments were conducted on several computers, average values are reported here. Note that the luminance of the white and red letters was similar, thus reducing the possibility that T_1 would mask T_2 more than distractor stimuli.

Procedure. On two thirds of the trials, a red target letter (T_1) appeared within the stream of white letters (T_1-present trials). On the remaining trials, no T_1 letter appeared (T_1-absent trials). T_1 could be one of three letters: an *H*, an *O*, or an *S*. The relative probability of the letters was varied, with a ratio of 1:4:9. Thus, one letter was presented nine times in 14 T_1-present trials, another was presented four times, and another was presented one time. The probability of each letter was counterbalanced across every 6 participants so that each possible assignment of each probability level to each letter was used once. Consequently, between participants, each letter occurred at each probability level equally often, and the probability manipulation was not confounded with letters. On all trials, a probe stimulus (T_2) appeared in the stream. On half of the trials, T_2 was an *X*, and on the remainder it was a *Y*.

Responses were made by pressing the assigned keys on a North American QWERTY keyboard, and RT to T_1 was recorded. The following keys were assigned as T_1 response keys: , < for *H*, . > for *O*, and /? for *S*. In the T_1-absent condition, participants were instructed to wait until the end of the stream of letters to press the space bar. This response was not speeded.

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At the end of the letter stream, a prompt for a response to T_2 appeared (*Did you see an X or a Y?*). The *X* key was used for an X response and the *C* key for a Y response. Participants used their left hand to respond to T_2 and their right hand to respond to T_1.

After each trial, feedback was given in the form of a plus or minus sign indicating to the participant whether the T_1 and T_2 responses made on the previous trial had been correct or incorrect. These feedback signs appeared adjacent to each other and were presented in the center of the screen. They also served as a fixation point for the next trial. The left sign was indicative of performance on T_1, and the right of performance on T_2. Each trial was initiated by pressing the space bar, which caused the fixation symbols to disappear and the next RSVP stream to begin 750 ms later.

At the beginning of the session, participants were required to read instructions regarding the experimental procedure. The instructions were then repeated verbally by the experimenter. The experiment began with two blocks of 32 practice trials each, followed by 672 experimental trials divided equally into eight blocks of trials. A complete crossing of the factors T_1 probability (T_1-present 1 4 9 or T_1-absent 7), T_2 letter (*X* or *Y*), and T_1–T_2 lag (1–8) consisted of 336 trials, or four blocks. In each complete crossing of all factors, the T_1 letter assigned to the Probability 1 condition was presented 16 times, each of the T_2 letters (*X* and *Y*) appearing once at every T_1–T_2 lag. The letter assigned to the Probability 4 condition appeared 64 times, each T_2 letter appearing 4 times at every T_1–T_2 lag, and the letter assigned to the Probability 9 condition appeared on 144 trials so that each of the T_2 letters was presented 9 times at every T_1–T_2 lag. Thus, T_1 appeared on 224 trials, according to the probability level assigned to the T_1 letter, and, in conjunction, each T_2 letter was presented at every T_1–T_2 lag. The T_1-absent condition (Probability 7) was tested on 112 trials. On these trials, each T_2 letter appeared 7 times at each T_1–T_2 lag. This complete crossing was presented twice throughout the experiment, resulting in a total of 672 trials.

The first task on each trial was to identify T_1 and respond as quickly as possible by pressing the appropriate response key. On T_1-absent trials, participants were instructed to wait until the end of the stream to press the space bar (unspeeded response). Following the letter stream, or after the space bar was pressed on T_1-absent trials, the prompt for a response to T_2 appeared. Participants were informed at the beginning of the experimental session that this response was not speeded. They were debriefed at the conclusion of the experiment.

Results

Individual data were checked for Task 2 accuracy to ensure that all participants could perform the task of identifying T_2 when T_1 was absent. Thirty individuals who failed to perform at or above 69% accuracy on Task 2 in T_1-absent trials were eliminated from further analyses, leaving 48 participants in the data set. In addition to the results reported subsequently, an analysis of Task 2 accuracy that included data for all participants ($N = 78$) was conducted. Although overall Task 2 accuracy was lower when all individuals were included in the analysis, effects of probability and T_1–T_2 lag, and the interaction between these variables, were similar to those observed in the analysis reported subsequently, in which participants performing below the Task 2 accuracy cutoff were excluded.

Analysis of Task 1 accuracy and RTs. Cell means were computed for Task 1 RTs and accuracy rates for each participant, probability level (1 4 9), and T_1–T_2 lag (1–8). Each cell for each participant was screened for outliers, which removed 2.5% of the correct responses (see Van Selst & Jolicœur, 1994). The cell means were entered into an ANOVA that revealed main effects of probability for Task 1 RT, $F(2, 94) = 118.7$, $p < .0001$, $MSE = 16{,}102.41$, and accuracy, $F(2, 94) = 23.9$, $p < .0001$, $MSE = 0.031$. Individuals were fastest ($M = 520$ ms) and most accurate ($M = .968$) on trials in which the probability of the T_1 letter was 9 and were slowest ($M = 661$ ms) and least accurate ($M = .880$) on the Probability 1 trials. On trials in which the probability of the T_1 letter was 4, RT ($M = 592$ ms) and accuracy ($M = .919$) were intermediate between the Probability 1 and 9 levels. There was no interaction of probability and T_1–T_2 lag and no main effect of T_1–T_2 lag for RT or accuracy in Task 1.

Analysis of Task 2 accuracy. Trials on which the response to Task 1 was correct were analyzed for accuracy on Task 2. For T_1-present trials, cell means for accuracy on Task 2 were calculated and entered into an ANOVA with probability (1 4 9) and T_1–T_2 lag (1–8) as variables. Results are shown in Figure 9.

In comparison with the accuracy rate on T_1-absent trials (.795), accuracy in identifying T_2 when T_1 was present was severely attenuated (.692), $F(1, 47) = 124.9$, $p < .0001$, $MSE = 0.009$. Figure 9 shows mean Task 2 accuracy rates for each T_1–T_2 lag and each probability level. In an analysis of T_1-present trials, a main effect of probability, $F(2, 94) = 21.3$, $p < .0001$, $MSE = 0.028$, indicated that, overall, a larger Task 2 deficit was evident for trials in which the T_1 letter was presented least frequently (Probability 1, $M = 653$) than for trials in which the T_1 letter was presented most frequently (Probability 9; $M = 732$). A main effect of T_1–T_2 lag showed that accuracy was most reduced at the earlier T_1–T_2 lags and improved steadily as T_1–T_2 lag increased, reaching performance equal to that of controls (T_1-absent trials) at T_1–T_2 Lag 7, $F(7, 329) = 36.6$, $p < .0001$, $MSE = 0.032$. An interaction

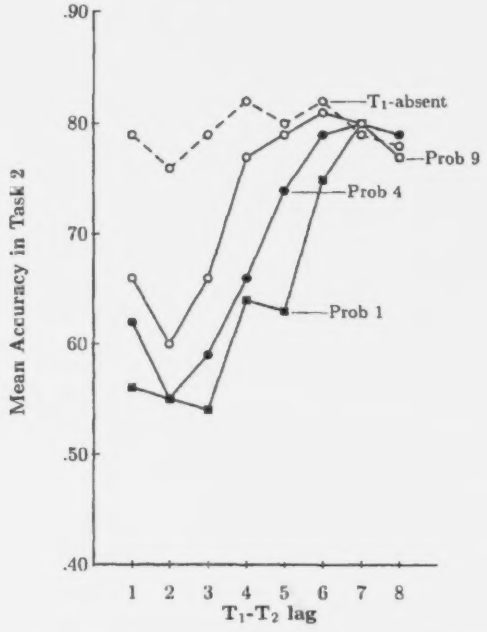

Figure 9. Experiment 4. Mean Task 2 accuracy in T_1-present and T_1-absent trials at each T_1–T_2 lag position. The effects of the probability (Prob. 1 4 9) of T_1 as a function of T_1–T_2 lag are shown. T_1 = Target 1, T_2 = Target 2.

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between probability and T_1–T_2 lag revealed that performance in the Probability 9 condition returned to be equal that of the control condition at an earlier lag position than the other two probability conditions, $F(14, 658) = 1.9$, $p < .026$, $MSE = 0.027$. The Probability 1 condition was slowest to return to performance equal to that of the control condition.

Analyses of the individual pairs of probability conditions showed that the interaction between probability and T_1–T_2 lag remained significant for the Probability 4 and 9, $F(7, 329) = 3.2$, $p < .0028$, $MSE = 0.011$, and Probability 1 and 9, $F(7, 329) = 2.4$, $p < .02$, $MSE = 0.033$, comparisons. This was not the case for the Probability 1 and 4 comparison, however, $F(7, 329) = 1.0$, $p > .45$, $MSE = 0.036$.

Task 2 as a function of RT_1. The central interference theory (Jolicœur, 1998) predicts that Task 1 RTs should correlate with accuracy on Task 2. When Task 1 occupies central mechanisms for a long period of time, the Task 2 deficit should be larger than when those mechanisms are occupied for a shorter period of time. Also, the duration of processing when central mechanisms are occupied should be reflected in RT_1. Consequently, long RT_1s should be correlated with poorer accuracy on Task 2, and short RT_1s should correlate with an increase in Task 2 accuracy, producing a smaller SAB effect. This prediction can be tested by examining the SAB effect as a function of RT_1.

However, one precaution must be taken here, because a correlation between RT_1 and T_2 accuracy could reflect a change in performance over time rather than a causal effect tied to central processing. For example, if performance on Task 1 and Task 2 improved as the experiment progressed, a relationship between RT_1 and T_2 accuracy might be observed, but for reasons other than those used to describe the prediction of the central interference theory. Individuals might be slower and less accurate at the beginning of the experiment simply because they are new to the task, and performance might improve over the course of the experiment as they become more proficient. Under these conditions, a correlation between RT_1 and Task 1 accuracy might be present, but only because slower responses made at the beginning of the experiment were coupled with poorer performance on Task 2 at the same point in the experiment. To deal with this issue, we examined the correlation between the size of the AB effect and RT within a subset of the trials over which there should have been little chance that performance could improve enough to produce a large correlation between RT_1 and accuracy in Task 2. This was accomplished by dividing the experimental trials into sections, representing progression through the experiment, and looking for a consistent SAB effect with a correlation between RT_1 and Task 2 performance across each section rather than across the experiment as a whole.

Each participant's data were divided into four sets of trials, each corresponding to 168 sequential experimental trials, resulting in a total of 672 trials. These trials were further divided into cells representing T_1–T_2 lag. The Probability 1 and Probability 4 conditions resulted in empty cells because of too few observations, thus, only the Probability 9 condition was examined. Each cell was subsequently divided in two, depending on whether RT_1 was above or below the RT_1 cell median for that participant. Mean accuracy in Task 2 for each cell was calculated and entered into an ANOVA with T_1–T_2 lag, short or long RT_1, and trial set (first, second, third, or fourth) as within-subject factors. The overall means for short and long RT_1s at each T_1–T_2 lag are shown in Figure 10.

The analysis revealed an effect of trial set, $F(3, 141) = 6.9$, $p < .0003$, $MSE = 0.046$. The mean Task 2 accuracy rate was lower in the first set of trials than in the other sets (Set 1, .70, Set 2, .74, Set 3, .74, and Set 4, .75). This effect was eliminated ($p > .54$) when the first set was not included in the analysis, thereby suggesting that performance on Task 2 was poorer on the initial 168 trials at the beginning of the experiment than throughout subsequent trials. This difference may indicate that participants should have been given more practice before the experimental trials. Most important, the correlation between RT_1 and Task 2 performance was consistent throughout the four sets of trials, providing support for the central interference theory. The pattern in the four trial sets was similar to that shown in Figure 10, long RT_1s produced a larger AB effect than short RT_1s.

Discussion

As expected, RTs in Task 1 were longer for stimuli presented less frequently than for stimuli presented more frequently. These results replicated the probability effect found in the previous experiments (Experiments 1, 2, and 3) and in the literature. Most important, the manipulation of signal probability in Task 1 also had a large effect on performance in Task 2. A larger Task 2 deficit was found for less frequent Task 1 stimuli than for more frequent Task 1 stimuli. Thus, the Task 1 manipulation of signal probability

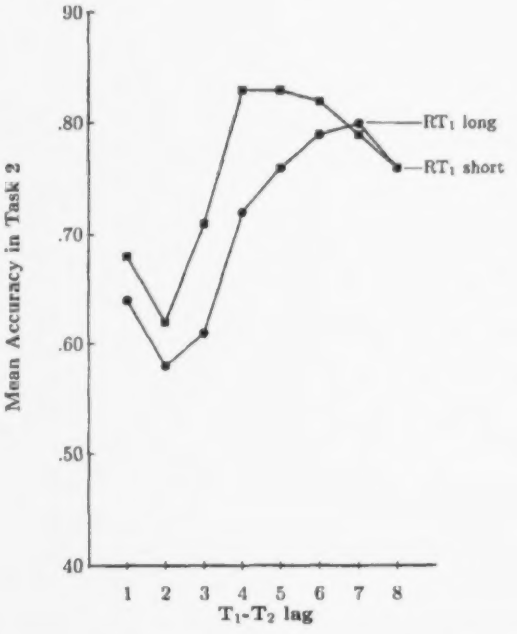

Figure 10. Experiment 4. Mean Task 2 accuracy depending on short or long response times (based on a median split of T_1-present trials only) in Task 1 (RT_1) as a function of T_1–T_2 lag. T_1 = Target 1, T_2 = Target 2.

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not only affected performance in Task 1 but carried forward to Task 2.

Such carry forward suggests strongly that the probability manipulation in Task 1 affected a stage that either coincided with the focus of dual-task interference (the bottleneck) or occurred before the locus of dual-task interference. Recall from Experiment 3, in which S_2 was masked, that we found an early effect of probability for the less frequent stimuli and a later effect for the higher probability levels. It is likely that the probability manipulation in this experiment also had an effect at more than one locus (one early and one late). The results of Experiments 1–3 suggest strongly, however, that effects of probability are entirely central, even when the stimuli are masked, when the manipulation ranges from moderate to high (that is, if we exclude the low-probability condition). Given that the dual-task deficit was modulated by a variation of probability in this range (i.e., across the Probability 4 and 9 conditions), it is likely that a longer period of central processing in Task 1 was responsible for the larger Task 2 deficit in the Probability 4 condition relative to the Probability 9 condition.

An alternative explanation with regard to the effect of probability in Task 1 should also be addressed at this time. It might be argued that an effect of probability could be found if participants were not performing the tasks according to the experimental instructions and were guessing on T_1 in an attempt to perform well on T_2. If individuals were guessing, the most frequent response would probably be the one associated with the highest probability T_1 letter, the Probability 9 condition. Thus, one might expect to see an effect of probability on T_1 due to a guessing strategy created to improve Task 2 performance. However, there is evidence in the results to suggest that this was not the case. First, if participants were guessing on T_1, and their most frequent response was the one associated with the highest probability T_1 letter, accuracy on T_1 should be high for the Probability 9 condition but low for the other conditions, including the T_1-absent condition. The results show, however, that T_1 accuracy rates were high across all probability conditions as well as when T_1 was absent (target absent, $M = .986$. Probability 1, $M = .880$, Probability 4, $M = .812$, and Probability 9, $M = 968$). In addition, if participants were guessing on T_1, one would not expect to see an effect of probability on T_2. Clearly, however, such an effect was present.

Experiment 5

Experiment 5 was the same as Experiment 4, except that the response to T_1 was unspeeded and deferred to the end of the trial. The goal was to discover whether the carry forward of the Task 1 probability manipulation observed in the SAB paradigm of Experiment 4 would also be observed in a more standard AB paradigm (deferred responses in both tasks). In addition, if the results changed markedly depending on whether Task 1 was speeded or unspeeded, this would provide valuable information concerning the locus of stimulus probability and of the locus of dual-task interference in the SAB and AB paradigms. Very similar results would suggest that the SAB and AB paradigms involve dual-task interference mechanisms that are affected in the same way by signal probability, which in turn would suggest a common source of dual-task interference in the underlying causes of the AB and SAB effects. Such a similarity would be consistent with the central interference theory (Jolicœur, 1998), which ascribes both effects to the postponement of the short-term consolidation of T_2 by concurrent central processing.

Method

Participants. Seventy-seven undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All of the participants had normal or corrected-to-normal vision.

Procedure. The method and procedure were the same as in Experiment 4, except that responses to T_1 were not speeded. Participants were instructed to wait until the end of the stimulus stream to make their responses to T_1 and T_2. The following prompt appeared at the end of the RSVP stream: *Did you see a red H, O, or S or no red letter?* After participants responded using the same response key assignment as in Experiment 1, a similar prompt was given for the T_2 response: *Did you see an X or a Y?* After making both responses, participants pressed the space bar to initiate the next trial.

Results

Individual data were analyzed for accuracy on Task 2 to ensure that all individuals could perform the task of identifying T_2 when no T_1 letter was present. Twenty-nine individuals who failed to perform at or above 69% accuracy on Task 2 in T_1-absent trials were eliminated from the data set. There were 48 participants in the final data set. As in Experiment 4, an analysis of Task 2 accuracy that included all participants ($N = 77$) was initially conducted. Again, accuracy on Task 2 was lower overall, but the effects of T_1–T_2 lag and stimulus probability were significant, as found in the analysis of Task 2 accuracy reported subsequently.

Analysis of Task 1 accuracy. Cell means were computed for Task 1 accuracy rates for each participant, probability level (1 4:9), and T_1–T_2 lag (1–8) and entered into an ANOVA. There was a main effect of probability, $F(2, 94) = 7.3$, $p < .0012$, $MSE = .025$, in that accuracy was superior in the Probability 9 condition ($M = .952$) and poorest in the Probability 1 condition (.908), with the Probability 4 condition (.926) falling between.

Analysis of Task 2 accuracy. Trials on which the response to Task 1 was correct were analyzed for accuracy on Task 2. As in Experiment 4, accuracy for Task 2 on T_1-present trials was severely attenuated (.726) in comparison with performance on trials in which T_1 was absent (.828), $F(1, 47) = 130.1$, $p < .0001$, $MSE = 0.015$. For T_1-present trials, cell means for Task 2 accuracy were calculated and entered into an ANOVA with probability (1 4 9) and T_1–T_2 lag (1–8) as variables. Results are shown in Figure 11.

Main effects of probability, $F(2, 94) = 35.8$, $p < .0001$, $MSE = 0.032$, and T_1–T_2 lag, $F(7, 329) = 17.5$, $p < .0001$, $MSE = 0.036$, were again evident, and an interaction between these variables approached significance, $F(14, 658) = 1.6$, $p < .075$, $MSE = 0.026$. A larger AB effect was observed for the T_1 letter associated with the lowest frequency (Probability 1, $M = .637$), and the smallest effect was seen for the Probability 9 condition ($M = .745$); performance in the Probability 4 condition fell between ($M = .70$).

The interaction between probability and T_1–T_2 lag was further examined by collapsing adjacent T_1–T_2 lag positions so that four positions (1–2, 3–4, 5–6, and 7–8) were entered into the analysis instead of eight. By this means, the effect of noise between T_1–T_2

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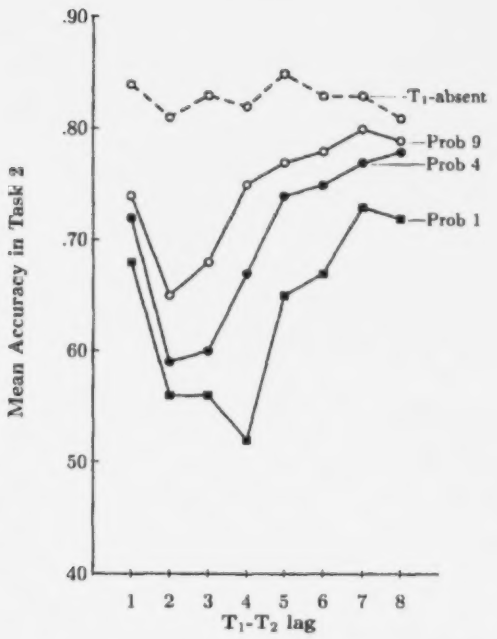

Figure 11 Experiment 5 Mean Task 2 accuracy for T_1-present and T_1-absent trials as a function of T_1–T_2 lag The effects of the probability (Prob. 1 4 9) of T_1 as a function of T_1–T_2 lag are shown T_1 = Target 1, T_2 = Target 2

lag positions was somewhat reduced, and the interaction between probability and T_1–T_2 lag became slightly more powerful. $F(6, 282) = 2.0$, $p < .062$, $MSE = 0.014$ This pattern of interaction replicates what was found in Experiment 4, and consequently the present results, although marginally significant in some analyses, nonetheless provide sufficiently strong converging evidence in support of an interaction between probability and T_1–T_2 lag This observation is bolstered by the fact that a separate analysis that included only the Probability 4 and 9 conditions produced a clear-cut interaction with lag, $F(3, 141) = 3.5$, $p < .016$, $MSE = 0.004$, as did an analysis including only the Probability 1 and 9 conditions, $F(3, 141) = 2.8$, $p < .040$, $MSE = 0.018$ However, the interaction was not significant when the analysis was restricted to the Probability 1 and 4 conditions, $F(3, 141) = 0.8$, $p < .466$, $MSE = 0.0189$ Why might this be so? It is important to remember that the Probability 1 condition included very few trials (relative to the Probability 4 and 9 conditions), by necessity Despite the relatively large number of trials in the experiment, there were few trials per participant and SOA in the Probability 1 condition, making participant means in this condition more variable than in the other two

Discussion

A comparison of Figures 9 and 11 reveals that the general patterns of results in Experiment 5 were very similar to those found in Experiment 4. In a separate analysis that compared Experiments 4 and 5 (with speeded vs. unspeeded Task 1 as a between-subjects factor), the interaction between T_1 probability and T_1–T_2 lag was highly significant, $F(14, 1316) = 2.6$, $p < .0011$, $MSE = 0.027$, whereas the three-way interaction among T_1 probability, T_1–T_2 lag, and experiment was not significant ($F < 1$). Furthermore, the results provided unequivocal evidence that the Task 1 manipulation of signal probability had a large and robust impact on Task 2 performance in that there was a very large main effect of T_1 probability in Task 2. Overall, the results are clear cut The effect of the probability manipulation in Task 1 had a marked carryover effect onto Task 2 that was similar across speeded and unspeeded versions of Task 1.

General Discussion

This research had two separate but interrelated goals. The first goal was to increase our understanding of effects of relative signal probability in simple speeded tasks This goal was pursued in Experiments 1–3 using the PRP paradigm, and the results and their implications have been summarized (see the general discussion of Experiments 1–3) The second goal was to use the knowledge gained in Experiments 1–3 to constrain the locus (or loci) of a dual-task interference effect known as the AB The aim was to situate a locus of the AB effect in relation to the PRP bottleneck by performing manipulations in both paradigms (AB and PRP) and by using methods of analysis providing constraints on the locus or loci of factor effects (Pashler & Johnston, 1989) Experiment 4, which involved an SAB paradigm (AB but with an immediate and speeded first task), served as a bridging experiment linking the PRP and AB experiments.

The relative signal probability of the first target in Experiments 4 and 5 produced large effects on Task 2 accuracy, regardless of whether Task 1 was speeded (Experiment 4) or unspeeded (Experiment 5) There were large and obvious carryover effects of the Task 1 probability manipulation on Task 2 performance In the PRP experiments (Experiments 1–3), the effect of the probability of S_2 was additive with SOA for unmasked stimuli, and we observed an underadditive interaction when S_2 was masked. Importantly, however, there were a range of probability levels for which the probability manipulation had additive effects with SOA. For this range of probabilities, the manipulation must have affected the duration of processing at the bottleneck or afterward, but not before. Furthermore, manipulations of probability in this range of probabilities produced unequivocal evidence for carry forward of the effect from Task 1 to Task 2 in the SAB and AB experiments (Experiments 4 and 5). As the probability of presentation of a T_1 letter decreased, accuracy in identifying T_2 decreased, thereby increasing the size of the deficit in Task 2. This effect of relative signal probability on Task 2 was evident in the SAB and AB experiments, that is, both when Task 1 was speeded and when it was unspeeded.

The finding that the probability of T_1 modulated the size of the AB suggests that the effect of probability was probably occurring at, or before, the locus of the AB effect The possibility that a variable can have an effect at a stage of processing subsequent to the locus of the AB effect and yet affect the size of the AB cannot be ruled out entirely, however. For example, startling the participant at the end of an experimental trial might result in loss of memory for T_2, thereby affecting accuracy of the response to T_2.

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However, such a retroactive effect seems very unlikely in the present context given the nature of the manipulation of interest (probability of the first target). Furthermore, all extant theories of the AB phenomenon are based on models of dual-task interference in which processing of T_1 influences the processing of T_2 in a proactive way (as opposed to a retroactive effect, Chun & Potter, 1995, Duncan et al., 1994; Jolicœur, 1998, Shapiro et al., 1994). In these theories, manipulation of a factor having an effect after the locus of AB interference would not be expected to affect the size of the AB. Postulating that manipulation of the probability of T_1 changed accuracy in Task 2 through a mechanism that had a locus after the stage, or stages, responsible for the AB effect would require us to reject all present models of the AB effect. We prefer to interpret the results as evidence that signal probability had a significant effect at, or before, a locus of processing causing the dual-task interference observed in Experiments 4 and 5.

Next, consider how the results from the PRP experiments can be used to constrain interpretation of the results from the SAB and AB experiments. To do so, we need to assume that the locus of probability effects was the same across all three paradigms. Given the importance of this assumption, we review the arguments that support it first. One of the most important considerations is the similarity of the tasks used to study probability effects in the PRP experiments and in the SAB experiment. In both cases, the same stimuli were used (the letters *H*, *O*, and *S*), and a speeded three-alternative discrimination was required. Also, the same probability manipulation was used in both cases (relative frequencies of 1 4 9). The results of Experiments 1–3 revealed that the viewing conditions of the stimuli were important as well. In particular, masking can induce an early locus of probability effects for low-probability targets. The viewing conditions across Experiments 3–5 were functionally similar, however, because the critical targets were shown for the same exposure duration and were preceded and followed by masking characters in all cases. Consequently, the results of Experiment 3, in which additive effects of probability and SOA were found for a range of probabilities (moderate and higher), are most likely valid for the conditions in place in Experiments 4 and 5. Thus, one main argument for the similarity of loci of effects across tasks hinges on the similarity of the tasks themselves. Given that the tasks were virtually identical across Experiments 3 and 4, it is not clear why the locus of effect of the probability manipulation was not the same.

Nonetheless, there were differences between the experiments. The RSVP streams were longer in Experiments 4 and 5 than in Experiment 3. Perhaps longer streams induce a greater shift toward an earlier locus than the shorter streams used in Experiment 3. At the moment, however, we have no evidence for this hypothesis. Furthermore, the results of Giesbrecht and Di Lollo (1998), as well as those of Brehaut et al. (1999), suggest that the addition of a single posttarget masking character is as effective in inducing AB effects as an entire stream of trailing characters. These results suggest that the short streams of Experiment 3 were probably functionally equivalent to the long streams of Experiments 4 and 5. Another possible source of difference across the PRP and SAB–AB experiments was the context in which probability was manipulated. That context was a three-alternative, speeded cross-modal choice task in the PRP experiments and a within-modality unspeeded choice task in the SAB and AB experiments. Finally, probability was manipulated in Task 2 of the PRP experiments but in Task 1 of the SAB and AB experiments. However, it is not clear why or how these differences would lead to a change in the locus of signal probability effects, and at the moment there is no evidence that these contextual differences could induce changes in the locus of probability effects. Thus, we are assuming pure insertion of the task subject to the probability manipulation in the two contexts, at least in terms of the locus of signal probability effects.

Next, consider the relationship between Experiments 4 and 5, in terms of the likely locus of signal probability effects in Task 1 of these experiments. These tasks were in all ways identical (stimuli, probability manipulation, and experimental context) with the exception that the response was immediate and speeded in Experiment 4 and deferred and unspeeded in Experiment 5. Again, however, it is not obvious why asking observers to make their response after a delay would shift the locus of probability effects to an earlier stage than the one affected by probability when their response could not be deferred.

Consequently, in the paragraphs to follow, we assume that the locus of signal probability was the same across tasks. This assumption is reasonable given the evidence available at the present time. However, new evidence from future research could overturn our conclusion concerning the invariant locus of probability effects across these tasks. In that case, we would need to reexamine the conclusions that we draw in the following concerning the locus of the SAB–AB bottleneck relative to the PRP bottleneck.

The results of the PRP experiments showed that the effect of the probability manipulation occurred either at the stage causing the PRP bottleneck or after (for moderate and higher probabilities). Consequently, the PRP bottleneck occurs either at or before the stage affected by signal probability. In Experiments 4 and 5, the effects of probability occurred either before or at the SAB–AB bottleneck. Thus, the SAB–AB bottleneck occurs either at or after the locus of the probability effect. Combining these two sources of information (as illustrated in Figure 12), and on the assumption that the locus of the probability manipulation was the same across tasks, logic dictates that a stage causing the SAB–AB bottleneck must either be coincident with the PRP bottleneck or occur later. Figure 12 helps illustrate this relationship.

Each diagram in the figure illustrates one possible combination of loci of probability effects in the PRP and SAB–AB paradigms. Probability affects a stage that is (a) either at the PRP bottleneck (left column) or after the PRP bottleneck (right column) and (b) either at the SAB–AB bottleneck (top row) or before the SAB–AB bottleneck (bottom row). Thus, using locus-of-slack logic and the possible loci for the probability effect to reference the locus of the dual-task bottlenecks in each paradigm, this analysis shows that one stage causing the SAB–AB bottleneck must be at least as late as the PRP bottleneck.

Jolicœur et al. (2001) provided converging evidence for this conclusion for the SAB paradigm. In their experiments, the number of stimulus and response alternatives was manipulated both in Task 2 of a PRP experiment and in Task 1 of an SAB experiment. The manipulation (number of stimulus–response alternatives) had additive effects with SOA in the PRP experiment, suggesting that the effects occurred at or after the PRP bottleneck. Furthermore, the SAB effect was larger when more stimulus–response alternatives were processed in Task 1. If we assume that the manipulation of number of stimulus–response alternatives influences the same stage (or stages) of processing when it is manipulated in Task 2 of

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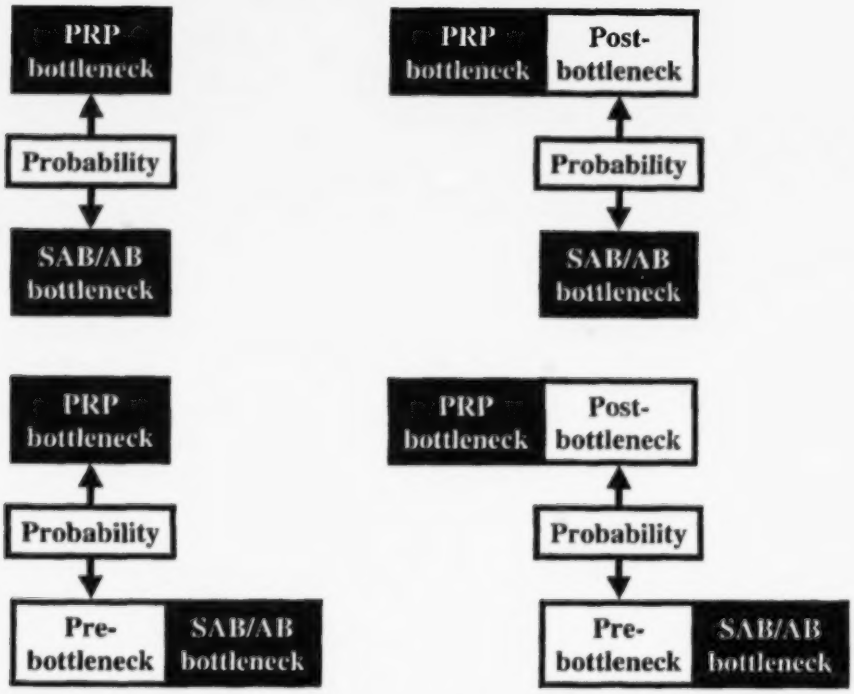

Figure 12 Four possible relationships between the locus of the SAB–AB bottleneck and the psychological refractory period (PRP) bottleneck based on constraints imposed by the locus of signal probability relative to these bottlenecks. The shaded areas represent the bottleneck stage(s) in the information-processing stream for each task. In all cases, the SAB–AB bottleneck occurs either at or after the PRP bottleneck. SAB = speeded attentional blink. AB = attentional blink.

a PRP experiment or Task 1 of an SAB experiment, then some of the SAB effect must originate at a stage of processing that coincides with the PRP bottleneck or at a later stage. Thus, the work of Jolicœur et al. (2001) has the same logical structure as the present work, and it provides strong support for the present conclusions concerning the relationship between the locus of the SAB and PRP bottlenecks. The present work focused on effects of signal probability rather than number of stimulus–response alternatives, allowing us to perform the manipulation in an unspeeded AB paradigm, something that was not possible for manipulations of the number of stimulus–response alternatives (see Jolicœur, 1999a). In the context of the central interference theory, both the AB and SAB effects have the same general cause: either a delay or slowing of a central stage required to perform Task 2 as a result of concurrent central processing required to perform Task 1 (Jolicœur, 1998; Jolicœur & Dell'Acqua, 1999). If accuracy in Task 2 decreases because of a period of waiting associated with the processing of the first target, then a longer period of waiting should result in a bigger deficit (see Arnell & Jolicœur, 1999; Chun & Potter, 1995; Jolicœur, 1998). This theoretical framework leads to the prediction that longer processing of T_1 should be associated with a larger deficit in Task 2 (in both the SAB and the AB paradigms). This prediction has been verified numerous times in the SAB paradigm, in which the duration of Task 1 processing can be assessed directly from RTs in that task (Experiment 4; see also Jolicœur, 1998, 1999a, 1999b, 1999c; Jolicœur & Dell'Acqua, 1999; Ross & Jolicœur, 1999). Because the tasks that were subject to dual-task interference in Experiments 4 and 5 were identical (i.e., Task 2), and because the patterns of results in these experiments were so similar (no interactions involving experiment), the results suggest that constraints on the locus of the SAB effect probably generalize to the AB. If so, the results of Jolicœur et al. (2001) provide converging evidence for a late locus of dual-task interference in the AB paradigm.

In the present SAB and AB experiments (Experiments 4 and 5), accuracy in Task 2 was higher at Lag 1 than at Lag 2. This pattern is sometimes found in AB experiments and has been referred to as Lag 1 sparing (Visser, Bischof, & Di Lollo, 1999). Potter, Chun, Banks, and Muckenhoupt (1998) proposed that a reduction in T_2 report accuracy with no Lag 1 sparing reflects two effects: task switching and AB. Task switching refers to an attentional switch between T_1 and T_2 that occurs when the characteristics of the target stimuli are very different, such as switching between modality or stimulus category (e.g., letters to Gabors), or when the task to be performed for each target is different. If the targets are similar enough, task switching is not

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necessary, and a U-shaped function across T_1–T_2 lag should be observed in the data. According to this view, monotonic effects of lag should reflect the combined effects of AB and task switching, whereas a U-shaped function with marked Lag 1 sparing should reflect a pure AB effect. Work reviewed by Visser et al. (1999) suggests that Lag 1 sparing and the AB phenomenon are independent of each other, the former resulting from the successful integration of T_2 with an input filter already configured for T_1. When two target stimuli are dissimilar, the input filter needs to be reconfigured for the second target, which results in poorest performance at Lag 1 and a monotonic function rather than a U-shaped function across subsequent lag positions. The AB effect, on the other hand, is a consequence of capacity limitations in short-term consolidation.

It is difficult to determine unequivocally whether the effects observed in Experiments 4 and 5 reflect only AB or some mixture of AB and task switching. If the latter is true, one could argue that the effect of probability that carried forward to Task 2 had a locus at some point after the AB locus but at, or before, a stage of task reconfiguration associated with task switching. We cannot completely rule out this possibility for the present results. If the AB already occurred before a task switch, however, it is not clear why effects of task switching should be confined to Lag 1, as suggested by the evidence reviewed by Visser et al. (1999). Their analysis suggests that the effects of attentional switching are restricted to Lag 1, and Lag 1 sparing is found when task switching is not a major component of the paradigm. In Experiments 4 and 5, we found Lag 1 sparing, along with effects of the probability of T_1 at several lags beyond Lag 1. Both of these results suggest that our paradigm primarily reveals effects of AB (rather than switching) and that probability had an effect at, or before, the locus (or one of the loci) of the AB effect. Furthermore, other work in our laboratory shows that carry forward of the probability manipulation in the first task can occur in a paradigm in which task switching could not influence the results. We used an AB paradigm in which Task 1 and Task 2 were identical, precluding any influence of task switching. Nonetheless, a manipulation of the probability of the first target carried forward and affected the magnitude of the AB.

Although it is conceivable, given our results, that the AB bottleneck could occur at a stage that is even later than the stage causing the PRP bottleneck, we do not believe that this alternative is very likely. It seems to us more probable that there is a stage of processing involved in both the AB and the PRP paradigms that contributes to, or constitutes, a processing bottleneck in both cases. One possibility for this stage is a late stage of stimulus categorization, which might be a prerequisite for both encoding into short-term memory (AB) and response selection (PRP). It is possible that the activation of an abstract code is a key function of the final process in encoding, immediately preceding response selection. Alternatively, the stage at which a name or abstract code is generated might constitute an initial substage of response selection. If we infer that the late locus of probability effects and the PRP bottleneck occur at a common stage and that code generation is an initial substage of response selection, this would certainly be in accordance with the findings from the PRP literature suggesting a bottleneck at, or just before, response selection (McCann & Johnston, 1992; Pashler, 1994b; Van Selst & Jolicœur, 1997).

The evidence provided by the PRP literature suggests a late locus for the PRP bottleneck. The present work suggests that at least one locus of AB interference in the AB and SAB paradigms is as late as (or later than) the locus of this bottleneck. The central interference theory (Jolicœur, 1998) provides a theoretical framework for the AB effect that postulates such a late locus of interference. The theory proposes that certain stages of processing require central mechanisms that are capacity limited and cannot occur in parallel. If central processors are occupied by Task 1, processing of Task 2 is delayed. The central interference theory also proposes that more than one central stage may be implicated in delaying Task 2, depending on whether the response to T_1 is speeded or unspeeded. When Task 1 is speeded, the stage of response selection delays short-term consolidation of T_2, and when Task 1 is unspeeded, short-term consolidation of T_1 delays short-term consolidation of T_2 (Chun & Potter, 1995).

One main difference between the SAB and AB paradigms is that some operations that can be deferred in the AB paradigm, because the response is made at the end of the trial, cannot be deferred in the SAB paradigm, because the response must be made as soon as possible after the onset of T_1 (Jolicœur, 1999a). From this vantage point, the present results suggest that effects of signal probability occur at a stage of processing that cannot be deferred in either paradigm. This suggests a locus such as a very late stage of categorization that would be required both for the short-term consolidation of a target (AB) and for response selection (for the common tasks in SAB and PRP). Perhaps this form of categorization is the initial phase of response selection, which would account for the additivity of the effect in Task 2 of the PRP experiments.

Conclusion

The most important conclusions from the present work were as follows. Effects of stimulus probability for unmasked stimuli were found to be additive with SOA in PRP experiments, indicating that the effect occurred at a stage of processing common to the PRP bottleneck or beyond it, and when the stimulus was masked, the effect was found to be underadditive with SOA, showing that signal probability also affected an early stage. However, even for masked targets, there were a range of probabilities for which the effects were additive with SOA, suggesting that for that range of values, the manipulation had an effect at or after the PRP bottleneck. The range-dependent interactions that we observed between stimulus quality and signal probability can explain the complex patterns of earlier results reported in the literature involving signal probability in choice tasks (see Figure 8). Stimulus probability in Task 1 also modulated the size of the AB effect, providing evidence that stimulus probability effects occur at or before a locus contributing to the AB effect. Importantly, the late component of the signal probability effect identified in the PRP experiments was shown to carry forward from Task 1 to Task 2 in the AB paradigm. On the assumption of an invariant locus of probability effects in the PRP and AB paradigms, the results indicate that a locus of the AB effect must be at least as late as the PRP bottleneck (Figure 12).

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